

Atlas and Manual of Dermatology and Venereology



Atlas and Manual of Dermatology and Venereology

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With 99 color plates and 73 black and white illustrations

This is a translation of 'Atlas und Praktikum der Dermatologie und Venerologie' by Professor Dr. W. Burckhardt published by Urban & Schwarzenberg. It has been translated and edited for the American market by Dr. Stephan Epstein.

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PREFACE TO THE AMERICAN EDITION

When I saw the proofs of Professor Burckhardt's *Atlas and Manual of Dermatology and Venereology* it occurred to me that a translation of such a book would be welcomed by American physicians. A combination of an atlas containing magnificent color pictures with a concise modern text, at a price which brings it within the reach of every physician, has no counterpart anywhere.

Professor Burckhardt's book is intended for the general practitioner and the nondermatologic specialists, especially allergists, dentists, internists, pediatricians, ophthalmologists, and medical students.

Emphasis is placed on the diagnosis and management of the more common diseases, and rare affections are only briefly noted. A new book offers the opportunity to eliminate antiquated methods of treatment and to concentrate on current therapy.

Professor Burckhardt is director of the Municipal Polyclinic for Skin and Venereal Diseases in Zurich, Switzerland. He is an internationally recognized authority on dermatology and has made numerous important contributions regarding its investigative and clinical fields. His *Atlas and Manual* have become so popular that within two years a second European edition has been published.

I have tried in my capacity as translator-editor to maintain the personal note of the book. Changes have been made to adapt concepts and therapy to American standards.

STEPHAN EPSTEIN

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A NOTE ABOUT THE ILLUSTRATIONS

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Dr. Rudolf Baer, Skin and Cancer Unit, Bellevue Medical Center, New York University, New York, New York (Figs. 20a and b).

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WALTER BURCKHARDT

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I ANATOMY OF THE SKIN

The skin is composed of an outer epidermis and an inner dermis or corium. Underneath the skin lies the subcutaneous fat.

The epidermis consists of several layers. At the bottom is the basal cell layer which is composed of cylindric basal cells. Since epidermal proliferation occurs in the basal layer it is also called the stratum germinativum. Small processes attach the basal cells to the corium. The wavelike surface formed by the papillae is a further aid in anchoring the epidermis to the corium. Melanocytes, the pigment forming cells, are found in the basal cell layer. Above the basal cells lies the prickly cell layer or stratum malpighii. The prickly cell layer is formed of round cells with large nuclei; the cells are joined together by means of intercellular bridges (prickles) hence the term 'prickly cell'. Fine fibrils passing through these bridges form a network binding the cells together.

In the upper part of the prickly cell layer the appearance of small granules signals the beginning of keratin formation. This portion is the keratohyaline layer or stratum granulosum. The cytoplasm is densely filled with dark granules. Above the stratum granulosum is a narrow homogenous zone without nuclei: the stratum lucidum. The high content of eleidin is responsible for its translucency. The stratum corneum is the uppermost layer of the skin. Here the nuclei have disappeared and the cells have cornified, but the cell borders can

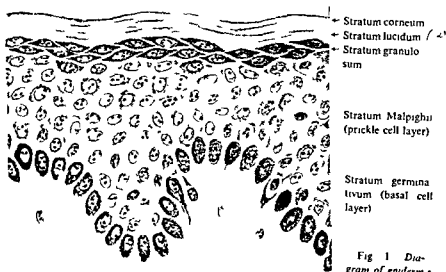


Fig 1 Diagram of epidermis

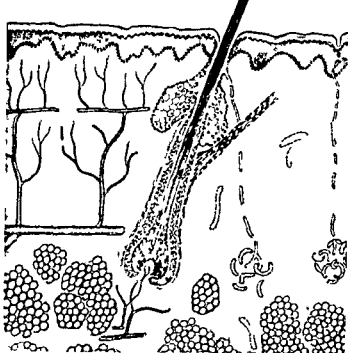


Fig 2 Diagram of epidermis, corium and appendages A hair follicle with its associated sebaceous glands and smooth erector pili muscle is shown At the base of the follicle are the hair bulb and hair matrix The eccrine sweat glands open onto the skin surface and the apocrine glands are connected with the hair follicles Blood vessels terminate at the papillae

still be seen in unstained preparations The stratum lucidum and the horny layer are most marked in the skin of the palms and soles

Blood vessels are found only in the corium, and not in the epidermis Although capillaries rise as far as the papillae, larger vessels such as arteries or veins are found only in the subcutaneous tissue Nerve fibers pass from the subcutaneous tissue into the corium and there branch to form a fibrillar network which ramifies within the epidermis as Langerhans' dendritic cells These fibrils are seen only in special silver or gold impregnated preparations In addition, certain specialized nerve endings called sensory corpuscles are located in the dermis

The appendages of the skin, i.e., hair and sebaceous and sweat glands, are also found in the corium The hair follicle is formed by an invagination of the epidermis which may extend down to the subcutaneous tissue At its base a rich network of blood vessels surrounds the hair bulb and hair generating matrix There are two types of sweat glands eccrine and apocrine Eccrine sweat glands are distributed over the entire body and open into the skin surface The secretory part of the gland lies between the corium and subcutaneous tissue, communicating with the surface by means of a corkscrew shaped duct The apocrine gland empties into a hair follicle Apocrine glands are essentially restricted to the axillae and the anogenital region A dense network of collagen, elastic, and reticular fibers provides the corium with the necessary strength and elasticity

II TYPES OF SKIN LESIONS

Careful examination of a dermatosis is important for accurate diagnosis. The patient should undress completely. Only in this way can the distribution of lesions be properly assessed. Also lesions may often be detected which had not been noticed by the patient.

The primary lesions are especially significant. Good illumination is essential. Use of oblique lighting as well as turning the patient will aid in bringing out surface detail. A magnifying glass may help. Cautious scratching preferably with a curette rather than with the fingernail reveals the nature of scales or crusts as well as of the underlying process. The intrinsic color of a lesion can be seen after blood vessels are emptied by firm pressure with a glass slide. One may describe skin lesions as being composed of new or primary and older or secondary elements.

Primary Lesions

The following are considered primary elements or lesions.

The Spot or Macule

This is a circumscribed change in color without any alteration of skin texture. It may be a localized redness caused by vasodilation (urtic roseola Fig. 145), a brown spot produced by increased pigment in the basal layer (Fig. 140), a blue discoloration from pigment deposits in the corium (blue nevus), or a white spot resulting from depigmentation (vitiligo Fig. 139).

The Wheal or Hive

A wheal consists of a somewhat raised, thickened, and whitish central portion and a surrounding reddish zone. Hives (urticaria, Figs. 98 and 99) result from edema within the corium. They generally form within 10 to 20 minutes and disappear after several hours. The whitish color is caused by edema compressing the blood vessels.

The Papule

This is a solid, circumscribed, definitely palpable and persistent thickening of the skin. Papules are usually markedly raised. Histologically they may be classified into three types. The first is produced by massive epidermal proliferation or acanthosis (basal cell carcinoma, wart Fig. 54). The second type results from inflammatory infiltrates within the corium (secondary lues, Figs. 150 and

151) In some papules both epidermal proliferation and dermal infiltration are present, these constitute the third type (lichen planus, Fig 65)

The Small Nodule

The small nodule is a special kind of papule. It is of pinhead size, projects as a hemisphere from the skin surface, and is definitely infiltrated. This is the primary lesion of eczema. Microscopically there is epidermal proliferation in which are found spongelike areas, the so called spongiotic edema. Another type of nodule is the polygonal, flat nodule of lichen planus. When nodules of either the eczema or lichen planus type become confluent, a diffuse lichenification of the skin results. Such lichenified skin is uniformly elevated and thickened with increased prominence of skin markings. Only histologic examination makes it possible to determine with certainty whether eczema, lichen planus, or some other disease produced the lichenification.

Nodule or Tumor

These terms are applied to infiltrated skin lesions larger than a cherry. Nodule is generally taken to mean a deeper infiltration, whereas tumor is used to describe those lesions that project above the surface.

The Vesicle and the Bulla

Effusions of serous fluid within or beneath the epidermis result in either small vesicles (dermatophytid, Fig 25, eczema, Fig 84), or large bullae (eczema, Fig 79, dermatitis herpetiformis Fig 58).

The Pustule

Accumulation of leukocytes within a vesicle converts it into a pustule. In this process the clear fluid becomes cloudy and finally assumes a yellow or greenish color (furuncle Fig 40).

Vesicles and pustules may arise at different levels in the epidermis. Subcorneal pustules lie just below the stratum corneum and elevate this layer (impetigo, Fig 36). The intraepidermal vesicle or pustule results in cleavage of the epidermis (pemphigus vulgaris). Subepidermal vesicles form between epidermis and corium (dermatitis herpetiformis Fig 58, erythema multiforme bullosum, Fig 59).

Secondary Lesions

The following are secondary lesions or elements.

The Hyperkeratosis

Hyperkeratosis is a simple increase in thickness of the horny layer. A callus (Fig 103) is a thickening of the stratum corneum produced by long standing



Fig 3 Red macule, due to circumscribed vasodilation

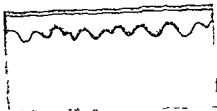


Fig 4 Brown macule, caused by localized increased pigmentation of the basal cell layer of the epidermis



Fig 5 Cutaneous papule formed by cellular infiltrate in the dermis ✓

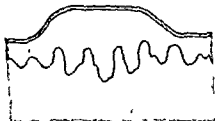


Fig 6 Epidermal papule, due to thickening of the epidermis — warts Basal Cell carcinoma

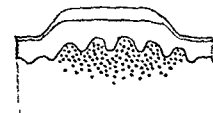


Fig 7 Mixed papule caused both by thickening of the epidermis and by cellular infiltrate in the dermis. (Urticaria planus)



Fig 8 Eczematous papule, caused by thickening of the epidermis and a spongiotic edema. There is also inflammation of the dermis and a parakeratotic horny layer

mechanical trauma Many inflammatory processes result in a localized, irregular and generally tenacious proliferation of the stratum corneum Examples are lichen planus verrucosus, the follicular hyperkeratosis of lupus erythematosus (Fig 62), and the irregular hyperkeratosis seen in carcinomas precancerous growths (Figs 117 and 118) and warts

The Scale

Normal skin undergoes continuous scaling or desquamation When skin has been encased in a plaster cast for several weeks the magnitude of this normal process becomes evident In inflammatory dermatoses there is often increased and defective cornification because the increased speed of cornification prevents

The Crust

The yellowish crust consists of dried secretions together with scales and leukocytes Crusts generally cover erosive lesions and must be removed to permit recognition of such underlying primary processes as eczema (Fig 93) carcinoma (Fig 124), or impetigo (Figs 36 and 37)

The Erosion

This is a superficial loss of tissue which does not extend below the epidermis

Excoriation is the term for traumatic erosions caused by scratching

The Ulcer

When tissue destruction extends below the epidermis and includes the skin appendages, the lesion is called an ulcer Ulcers heal with scar formation Ecthyma (Fig 37b) stasis ulcers (Fig 114) and carcinoma (Fig 124) are typical examples

Atrophy and Scar Formation

Atrophy is heralded by a thinning of epidermis and corium accompanied by loss of the follicles and sebaceous and sweat glands the easily be picked up absence of hair are also characteristic Circumscribed atrophy is common after certain inflammatory diseases, among which are lichen planus lupus erythematosus (Fig 61)

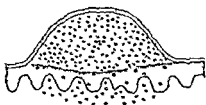


Fig 9 *Subcorneal pustule* The stratum corneum is separated from the underlying skin by an accumulation of edema fluid and leukocytes. Leukocytes have infiltrated the corium and are migrating through the epidermis.



Fig 10 *Subepidermal vesicle* The fluid has lifted the entire thickness of epidermis and the corium contains an inflammatory infiltrate.



Fig 11 *Intraepidermal vesicle* Acantholysis has produced an irregular fluid-filled space in the epidermis in which leukocytes and detached epithelial cells are found.



Fig 12 *Erosion* A shallow defect, limited to the epidermis, partially filled with serum and leukocytic debris.

Fig 13 *Ulcer* The tissue defect extends into the corium. The entire epithelium and its appendages have been destroyed. A collection of serum and debris forms the base, while the adjoining corium shows an inflammatory infiltrate.



lupus vulgaris, and tertiary lues (Fig 153) In certain diseases atrophy of the epidermis coexists with hypertrophy of the corium (scleroderma Figs 76 and 77) When a tissue defect caused by ulceration is incompletely repaired a scar results Only a thin epidermis is produced, with no papillae and with a flabby dermis Occasionally, however, a keloid like thickening develops Pin point scars occur after circumscribed necroses as in folliculitis furuncles herpes zoster, acne

Polymorphous Lesions

The lesions of many skin diseases have several components Psoriasis consists of a macule covered by scales, impetigo is a crusted erosion syphilitic condylomata are eroded papules, the lesions of erythema multiforme may be a papule with a blister in the center surrounded by an erythematous halo Eruptions may be monomorphic lichen planus composed of similar papules fits this category but more frequent are polymorphic eruptions In scabies for example, pustules papules, excoriations and crusts may all be present

Location, Configuration and Extension

An eruption may be limited to a small area or it may be extensive In the latter case it is called an exanthem If an eruption is present upon a mucous surface, the term enanthem is applied Lesions may be found only in certain areas or they may involve the entire integument The regional distribution of lesions is often of diagnostic help Certain dermatoses such as psoriasis favor the extensor surfaces of arms and legs whereas atopic dermatitis is most pronounced on the flexural side of joints When external influences are involved as in light dermatitis the face ears hands and other exposed parts will be chiefly affected

In contact dermatitis the area of contact with the causative agent usually is the first to break out The localization of some dermatoses is explained by anatomic variations of the skin Certain dermatophytes grow most luxuriantly on the thick horny layer of palms and soles whereas other fungi such as trichophyton prefer hair and consequently affect the hairy parts of the body Sebaceous glands are most numerous on the face back and chest and these are consequently the sites favored by acne A segmental distribution as in herpes zoster, implies spinal or cranial nerve involvement

Many diseases show lesions with a characteristic configuration Localized infections caused by fungi or pyogenic cocci often show a progressive centrifugal growth Gradually developing immunity leads to central healing and ring shaped lesions (Figs 27 28 and 30) Rings may fuse to yield polycyclic or

serpiginous formations. However, lesions of this type may also be produced by a variety of diseases of unknown etiology, presumably infections as well as by certain blood borne infections such as tertiary lues, the ring shape in these diseases suggests local immunologic processes

S. l. diseases & atrophy - 6 p. v. d. p. u.
 1. l. p. u. l.
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apparently normal skin of rabbits, the normal bacterial flora of the skin may become virulent. The human organism also, especially the skin, harbors such '*microbes de sortie*' they cause disease only when the resistance of the skin has been lowered.

Infectious Allergy

The antibodies which are elicited by an infection may destroy the causative organisms and neutralize their toxins, very often however, other substances are also produced which lead to inflammatory reactions. These antibodies aggravate or actually cause the symptoms of the disease. The phenomena occurring in the course of an infection have been described by von Pirquet he called them 'allergy' meaning an altered reaction.

Von Pirquet selected as a special example tuberculosis. An organism which has not been infected with tuberculosis does not react to tuberculin. The tuberculin reaction becomes positive only several weeks after the infection, when sensitizing antibodies are present. In infectious diseases the sensitizing factors are always combined with curative, neutralizing antibodies. Infectious allergy is part of the immunologic process. A positive tuberculin reaction permits the conclusion that there is a certain, though usually incomplete immunity against tuberculosis. The conditions in mycoses are similar, here also an infectious allergy develops, with a positive trichophytin reaction. The parallelism between allergic and immunizing processes is especially evident in fungus infections. In inflammatory ringworm with a strong trichophytin reaction there is a tendency for spontaneous cure and for development of a lasting immunity, whereas tinea caused by *microsporon* fungi lacks infectious allergy and clears up only very slowly.

Infectious allergy produces hypersensitivity to vaccines from the causative organism manifested by delayed inflammatory reactions which occur in the dermis. They are called tuberculin type delayed reactions. These tests have a certain diagnostic value (Table 1).

Table 1
Skin Reactions in Infectious Diseases
1 Reactions Due to a Toxic Effect

Disease	Reactions	Course during infection
Diphtheria	Schick test (delayed reaction)	Positive in susceptible persons Negative during and after infection
Scarlet fever	Dick test (delayed reaction)	Positive in susceptible persons Negative during and after infection

III. INFECTIOUS DISEASES OF THE SKIN

Protection of the Skin against Infection

Natural Resistance

Immunobiology deals with the resistance of the skin against microbes of all kinds, as well as against the poisons of insects spiders and worms. The bactericidal effect of the horny layer and of the secretions of the skin, such as sebum and sweat, are elements of the natural resistance, the surface of the skin is acid and has an average pH of 5.3. The skin maintains this "acid mantle", its buffering capacity also plays a role in the protection against chemical insults.

Immunity

Active or latent infections produce specific antibodies, and in this way often cause an absolute or relative immunity. Immunity may last for many years after general infections which mainly affect the skin, for example, variola, vaccinia and chicken pox. A similar lasting immunity follows herpes zoster, inflammatory ringworm and pityriasis rosea, these are diseases which one usually gets only once. With syphilis, gradual but incomplete stages of immunity develop, this is the reason for the polymorphic clinical picture of syphilis and its various stages. Pyogenic organisms, such as staphylococci and streptococci, produce immunity against the individual strains, therefore, the skin is usually healthy although it is infested by numerous pyococci. Pyodermas occur only when foreign strains of cocci invade the skin, or when the virulence of the existing pyococci is increased or when the resistance of the host is diminished. Immunity develops also against the poisons of insects.

Immunobiologic Disbalance as Cause of Skin Diseases

The immunologic balance may be unfavorably influenced by disturbances of the general health, *by physical or emotional overexertion*, by debilitating diseases such as pneumonia, malaria, typhoid fever, measles, carcinoma, avitaminosis, malnutrition, also by potent drugs, such as corticosteroids, antibiotics, sulfonamides, arsphenamines. In this way latent infections may be activated. Examples are the occurrence of herpes zoster or erythema multiforme following fatigue, an eruption of herpes simplex during pneumonia, a cold, or after exposure to the sun, the development of erythema nodosum following sul }

exists in animal experimentation which seems to be the same



Fig 14 *Lupus vulgaris* Grouped lupus nodules of the cheek

2 Reactions Due to Infectious Allergy

Disease	Reactions	Course during Infection
Tuberculosis	✓ Tuberculin test (delayed reaction)	Negative in noninfected persons Positive during and after infection
Dermatomycosis	✓ Trichophytin test	Negative in noninfected persons Positive during and after infection
Lymphogranuloma venereum	<u>Frei test</u>	Negative in noninfected persons Positive during and after infection
Chancroid	Ito-Reenstierna test	Negative in noninfected persons Positive during and after infection

There is an ambivalence of the immunizing processes, after a patient has recovered from a disease we may find either a disappearance of previously positive reactions or the development of a positive intracutaneous reaction. In the former case primary toxins, which normally caused an inflammatory reaction, are neutralized by antibodies, in the latter instance we are dealing with allergic processes. Minor skin eruptions, usually of short duration, caused by hematogenous spread from a microbic focus, are called microbids, or "ids", examples are tuberculids, bacterids, trichophytids, or dermatophytids. It has not been decided whether they are mainly due to a deficient development of immunity or to increased infectious allergy //

Tuberculosis of the Skin

Differentiation must be made between true tuberculosis of the skin and tuberculids. In the former, the diseases are progressive, whereas the tuberculids are microbids of relatively brief duration and benign character.

True Tuberculosis of the Skin

✓ Primary Complex ✓

The tuberculous primary complex of the skin is rare, it consists of a slightly infiltrated, soft ulcer with undermined edges and a regional lymphadenitis. The primary complex is found on the face, the hands, the mucous membrane of the mouth, and occasionally on the genitalia. It is caused by direct infection from sputum, pus or urine from tuberculous persons or from food such as milk from tuberculous cows. Tubercle bacilli are abundant in the ulcer with the change of the immunologic status the primary complex appears more like lupus vulgaris.



Fig 15 *Lupus vulgaris*
Indurated brownish red le-
sions of the posterior fold of
the axilla. The brownish color
is more apparent when pres-
sure is applied with a glass
spatula.



Fig 16 *Tuberculosis ver-
rucosa cutis*. Warty lesions
on an inflamed indurated
base. The scarred depigmen-
ted part in the center is the
result of previous local
treatment with caustic agents.

Lupus Vulgaris (Figs 14 and 15)

Lupus vulgaris is the most frequent form of tuberculosis of the skin. It begins as grouped, brownish red slightly scaly papules from pinhead to coin size. Under pressure with a glass slide the light brown so called "apple jelly" color of the tuberculous granulation tissue is seen, a metal probe, pushed through the thinned epidermis, easily enters the soft lupus tissue. The individual papules may coalesce to form larger plaques, occasionally very prominent papules or nodules are formed (lupus tumidus). The most frequent site of lupus vulgaris is the face, but it may occur on any part of the body, including the mucous membranes of the mouth and nose. The lesions of lupus vulgaris may be ser-piginous and tertiary lues must be considered a diagnostic possibility in these cases. Without treatment lupus vulgaris grows slowly through the years to involve large areas of the skin. The tuberculous granulation tissue attacks and destroys the underlying tissues such as the cartilage of the nose, ears and eye lids, which may be mutilated by ulceration and scarring.

Histologically typical, nodular, tuberculoid lesions are found and tubercle bacilli can be demonstrated in the sections as well as by animal inoculation. Lupus vulgaris is caused by hematogenous dissemination of bacilli from a tuberculous focus, or by direct extension or lymphogenous spread from tuberculosis of lymph nodes. Accordingly, lupus vulgaris either remains a rather local condition or becomes more diffuse.

Tuberculosis Verrucosa Cutis (Fig 16)

Tuberculosis verrucosa cutis is caused by superinfection of a previously infected individual. Examples are people with tuberculosis of the lung who infect themselves with their own sputum, butchers and farmers who repeatedly handle tuberculous cattle and the attendants in postmortem cases who are infected by contact with tuberculous organs (prosector's wart).

Tuberculosis verrucosa cutis consists of red papules with a warty surface which may become confluent to form larger plaques.

Histologically there is hyperkeratosis and a thickening of the epidermis, in the cutis areas of central necrosis are surrounded by a tuberculous infiltrate consisting of giant cells epithelioid cells and peripheral lymphocytes. Tubercle bacilli can be demonstrated in histologic sections or by animal inoculation.

Scrofuloderma (Tuberculosis Cutis Colligata)

Scrofuloderma is a disease of the cutaneous and subcutaneous tissues often originating from a neighboring tuberculous lymph node. The overlying skin

← Fig. 17a *Papulo necrotic tuberculid* Red papules with necrotizing central pustules
The necrosis is followed by a small crusted ulcer which forms a scar



~ Fig. 17b *Papulo-necrotic tuberculid* Bes des roundish papules there is a larger
les on with an ulcer in the center which was preceded by necrosis

and sometimes it is fixed to the underlying lymph node or bone. Thin pus, which may contain tubercle bacilli, is discharged from fistulae. The usual site is the neck.

Rare Forms of Tuberculosis of the Skin

Rare forms of tuberculosis of the skin are ulcerative tuberculosis of the newborn following infection after ritual circumcision, and ulcerative skin tuberculosis of cachectics. In both instances a severe septic condition occurs because immunity either has not yet developed or has disappeared. Lupus miliaris faciei shows a dissemination of small papules on the face, its course is benign, similar to that of a tuberculid.

Tuberculids

Papulo-Necrotic Tuberculids (Figs 17a and b)

the individual lesions heal with small starlike scars, while new papules may appear. Histologically there is tuberculoid granulation tissue surrounding a central necrosis.

Lichen Scrofulosorum

eruptions, on the gluteal region, and occasionally on the trunk and face. Spontaneous cure within a few weeks is the rule. Histologically there is a tuberculoid granuloma directly below the epidermis.

Erythema Induratum (Fig 18)

Erythema induratum of Bazin is found on the posterior surfaces of the lower limbs. It appears in the form of coin sized a bluish red discolora- tions. Older ones of a more bluish red color and healed lesions with a persistent brownish discoloration. Some nodules ulcerate and turn into deep punched out ulcers which heal with scars during the course of several weeks. Erythema induratum has a great tendency to recur and may persist for years. Histologically there are perivascular inflammatory foci in the cutis and subcutis which often show tuberculoid structure. Frequently a diagnosis is made possible only by a general examination of the patient which may reveal tuberculosis of some other organ, or by the therapeutic effect of specific antitubercular treatment.

Fig 18. Erythema Induratum (Bazin). Subcutaneous nodes on the flexor side of the lower leg, bluish red discoloration of the skin

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Tuberculids

Papulo-Necrotic Tuberculids (Figs 17a and b)

Papulo necrotic tuberculids have a predilection for the extensor surfaces of arms and legs the gluteal region and the face. The eruption consists of crusted papules which later become small ulcers. During the course of several weeks the individual lesions heal with small starlike scars while new papules may appear. Histologically there is tuberculoid granulation tissue surrounding a central necrosis

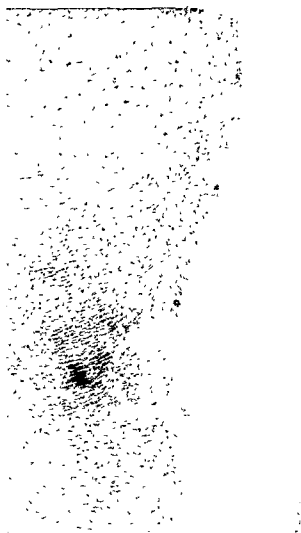
Lichen Scrofulosorum

Lichen scrofulosorum appears as a crop of roundish, flat, pinhead sized papules of a bright or pale red color which may become confluent and form coin sized to palm sized plaques. They occur on the extensor surfaces of the extremities on the gluteal region and occasionally on the trunk and face. Spontaneous cure within a few weeks is the rule. Histologically there is a tuberculoid granuloma directly below the epidermis

Erythema Induratum (Fig 18)

Erythema induratum of Bazin is found on the posterior surfaces of the legs in the form of coin sized, deep seated cutaneous nodules the overlying skin shows a bluish red discoloration. Besides relatively fresh bright red nodules there are older ones of a more bluish red color and healed lesions with a persistent brownish discoloration. Some nodules ulcerate and turn into deep, punched out ulcers which heal with scars during the course of several weeks. Erythema induratum has a great tendency to recur and may persist for years. Histologically there are perivascular inflammatory foci in the cutis and subcutis which often show tuberculoid structure. Frequently a diagnosis is made possible only by a general examination of the patient which may reveal tuberculosis of some other organ or by the therapeutic effect of specific antitubercular treatment

Fig 18. Erythema Induratum (Bazin). Subcutaneous nodes on the flexor side of the lower leg; bluish red discoloration of the skin.



cure, and treatment should be continued 2 months beyond the time the lesions have cleared up clinically. When symptoms of intolerance occur, for example nausea, vomiting, or purpura, the dose must be reduced or the drug must be discontinued.

Vitamin D₂ therapy may be combined with isoniazid, inasmuch as vitamin D₂ has a relatively nonspecific effect and isoniazid is a specific antitubercular drug. Recurrent lesions may be treated with PAS, streptomycin, or a combination of PAS and isoniazid. General measures such as a stay in a sanitarium and irradiation with natural sun or ultraviolet light are indicated in cases of extensive lupus vulgaris and scrofuloderma and in the recurrence of erythema induratum. These measures now have become unnecessary in milder cases of lupus vulgaris, in tuberculosis verrucosa cutis, and in tuberculids.

Ultraviolet irradiation. Two mercury arc lamps are used for irradiation of the whole body. The distance from the lamp is 1 m. Depending on the intensity of the burner, the initial dose is 1 to 2 minutes for each side of the body, a dose which should produce only a mild erythema. Each exposure is increased by 1 minute, until 15-minute periods are reached. A total of 20 or 30 irradiations two or three times a week is recommended. Then treatment is discontinued for 2 months so that the skin may lose its increased resistance to ultraviolet rays, due to the thickening of the horny layer. The eyes must be shielded during treatment.

Boeck's Sarcoid (Sarcoidosis, Lupus Pernio, Lymphogranulomatosis Benigna) (Fig. 19)

Some authors consider sarcoidosis a separate disease entity, others believe it to be a special form of tuberculosis. Transitions from one to the other have often been observed.

Boeck's sarcoid occurs on the skin in two distinct forms. In one there are lupus vulgaris-like papules in which the brownish color of the granulation tissue is revealed by pressure with a glass slide. The papules are often arranged in a ringlike lesion. In the other form of sarcoidosis there are subcutaneous nodules which may become attached to the skin. These nodules are found on the lower legs and hands and, in addition, on the trunk, arms and forearms, a characteristic infiltrate has been described. The histologic picture of the gran is surrounded by epithelioid cells are occasionally found and necrosis is absent.

Sarcoidosis is usually a systemic disease with epithelioid cell infiltrates occurring in many organs. Frequently the spleen and lymph nodes, especially the

Nonspecific perivascular inflammations with the same clinical picture respond better to penicillin or broad spectrum antibiotics

Doubtful Tuberculids

A doubtful tuberculid is the rosacea-like tuberculid (see "Rosacea", page 205) Erythema nodosum (page 60) has some relationship with tuberculosis but is not necessarily a tuberculid

Associated Manifestations of Tuberculosis

In all cases of tuberculosis of the skin a search must be made for tuberculosis of other organs by means of x-ray examination of the lungs, by urinalysis and by examination of the mucous membrane of the nose. It is strange that patients suffering from advanced tuberculosis of the lungs, kidneys or bones usually do not show skin involvement, whereas relatively minor infections of the lymph nodes are often found as the origin of tuberculosis of the skin. Occasionally tuberculids precede new manifestations in visceral organs

Therapy of Tuberculosis of the Skin and Tuberculids

Formerly treatment of tuberculosis of the skin consisted of caustic ointments destructive electrosurgical procedures, and irradiation with the Finsen or Kromayer lamps. This treatment was replaced first by the vitamin D therapy (Charpy, Dowling), and more recently by chemotherapy with streptomycin, isoniazid, and *p*-aminosalicylic acid (PAS). Lupus vulgaris has lost its terror; it can usually be arrested and cured. However, treatment still has to be continued for months or years and therapeutic courses must be repeated; therefore, patients with lupus vulgaris, as with any other form of tuberculosis, must be kept under constant supervision.

Vitamin D₂ (calciferol) treatment * First week, three times weekly 600 000 units of vitamin D₂ in alcoholic solution (15 mg of calciferol), second to fourth weeks twice weekly 600,000 units of vitamin D₂, fifth to 52nd or 76th weeks once weekly, 600 000 units of vitamin D₂

Since the elimination of large quantities of calcium puts a strain on the kidneys, regular checks of nonprotein nitrogen and blood calcium levels are necessary.

Treatment with isoniazid (isonicotinic acid hydrazide). Initially 5 mg of isoniazid per kg of body weight are taken daily; later 8 to 10 mg daily. The daily dose ranges from 300 to 500 mg. It usually takes 6 to 10 months for a

* In Great Britain Sterogyl 15 is used (ampules of alcohol solution for oral administration made by Roussel, each containing 600 000 units of vitamin D₂) or calciferol tablets B N F (each containing 50 000 units) are given twice daily.

hilar nodes of the lungs, are enlarged X ray of the lungs often shows fine miliary densities Ostitis fibrosa cystica, with spindle shaped swellings of the bones, may occur, especially in the fingers and toes. Chorioiditis and parotitis also have been observed The tuberculin reaction is usually negative, an intradermal test with an extract from a lymph node of a patient suffering from sarcoidosis produces a slight local inflammation after several weeks (Kveim test)

The course of sarcoidosis is usually chronic, but benign and spontaneous cures have been observed Gradual progression of the pulmonary changes also occurs with resulting dyspnea and increasing cachexia

Treatment Sarcoidosis is rather resistant to treatment but vitamin D₂ or isoniazid are given as in tuberculosis of the skin and cortisone has been beneficial in some instances In severe cases chemotherapy should be combined with a stay in a sanitarium

Leprosy (Hansen's Disease)

(Figs 20a and b)

There are more than a million people suffering from leprosy in the world and some believe the figure to be nearer to five million Although it is still one of the most common infectious diseases it is rare in the United States and still more unusual in Western Europe

The causative organism is an acid fast bacillus *Mycobacterium leprae* The infection occurs chiefly early in life, the incubation period probably may last several years The first symptom of the disease often is a pale, hypopigmented spot (macula alba) in which cutaneous sensation sooner or later is disturbed, an injection of histamine will provoke a wheal but no red flare Later in the course of the disease two principal or "polar" forms are distinguished

1 Lepromatous leprosy consists of papules and nodules especially located on acral parts—face, ears elbows buttocks, etc These lesions are called lepromas and contain numerous lepra bacilli The nasal mucous membrane is often involved with ulceration and perforation or destruction of the septum Acid fast bacilli are abundant in the nasal secretion in about one-third of early cases and in all advanced cases of this type Without treatment this form often terminates fatally in 12 to 15 years

2 Tuberculoid leprosy, on the other hand is a relatively benign, generally self limited form of the disease There are flat, hypopigmented macules and also tuberculoid often circinate plaques resembling Boeck's sarcoid both clinically and histologically Nerve involvement is always demonstrable, and may be the most conspicuous feature of tuberculoid leprosy, although it is frequently impossible to find bacilli



Fig 19. *Sarcoidosis (Boeck's sarcoid)* Subcutaneous nodules of the fingers.

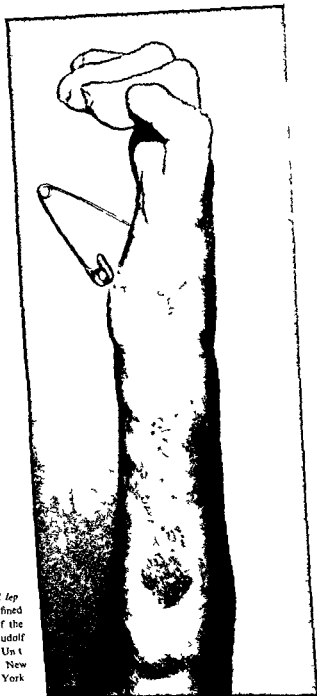


Fig. 20b *Tuberculous leprosy* showing sharply defined lesions and anesthesia of the skin (Courtesy of Dr. Rudolf Baer, Skin and Cancer Unit, Bellevue Medical Center, New York University, New York).



Fig. 701. *Leprosy* (Courtesy of Dr. Rudolf Baer, Skin and Cancer Unit, Bellevue Medical Center, New York University, New York, New York.)

Mycoses

Mycoses are caused by *Fungi imperfecti*. There are two main groups of fungal diseases of the skin: (1) the dermatophytoses which primarily attack the epidermis and its appendages, for example trichophytosis, epidermophytosis, favus, erythrasma, pityriasis-versicolor and moniliasis, and (2) the deep mycoses of the subcutaneous tissue where the skin is involved secondarily, as in actinomycosis, sporotrichosis, coccidioidomycosis, blastomycosis and madura foot.

Superficial Mycoses

In the superficial dermatomycoses the causative organism is found by direct examination of scales, vesicles, pustules and hair which are made transparent by the use of xylol (Fig. 21) or a 10 to 20 per cent potassium hydroxide solution. For definite identification of the causative fungi, cultures are necessary, but for practical purposes a microscopic examination is sufficient. The diagnosis may be supported by intracutaneous tests with fungus vaccines such as trichophyton, sporotrichum or coccidiomycin, which are positive in the inflammatory forms of ringworm.

The clinical picture to a certain extent depends on the virulence of the causative organism. Very virulent fungi produce deep suppurative inflamma-



Fig. 21. Fungal hyphae in scales made transparent by xylol.

A group of macular cases is designated "indeterminate" because they cannot, at present, be classified as either lepromatous or tuberculoid leprosy, and may develop into either.

The lepromin or Mitsuda test is carried out with a sterile suspension of ground-up lepromatous granulation tissue, rich in bacilli. It is read after a period of 2 to 3 weeks following the intradermal injection. The reaction is negative in the lepromatous form, in which it signifies a lack of defense against the bacilli. A positive lepromin reaction is found in the tuberculoid type and in about half of nonlepromatous persons, it is an inflammatory nodule, often ulcerated, and indicates resistance to the disease and a favorable prognosis.

Treatment. Chemotherapy with sulfone drugs such as Promin, Promizole, Promacetin, and the parent sulfone, diaminodiphenyl sulfone (Arlosulfon), has changed the fate of patients suffering from leprosy, all tuberculoid and early lepromatous cases—and most late ones—now can be cured. Ulcers heal rapidly but in general the effect of treatment is slow and in lepromatous cases 2 to 5 years of treatment, or more, are required.

Most countries have a policy of rigorous isolation for bacteriologically positive cases. Regular examinations for activity are required of bacteriologically negative cases.

Cutaneous Leishmaniasis (Oriental Sore)

The causative organism, *Leishmania tropica*, is transmitted to man by the bite of an infected sand flea. After an incubation period of 1 to 4 weeks, or more, papules occur mostly on exposed parts of the body and ulcerate as time goes on. Spontaneous cure with scar formation within a year is the usual outcome, but occasionally there occur lupus vulgaris—like infiltrates and ulcerations which may lead to mutilation of the face and hands. The causative organism may be found in smears. A leishmanin reaction, similar to the tuberculin reaction, may facilitate the diagnosis.

Treatment. Local destruction is carried out by electrodesiccation or with caustic ointments, such as 20 per cent pyrogallol in Vaseline. Antimony preparations are given systemically.

Oriental leishmaniasis occurs in the countries adjacent to the eastern Mediterranean and the Black Sea. American leishmaniasis, prevalent in South America, is a more severe disease which attacks the mucous membranes of the mouth and nose in addition to the skin. Its cause is *Leishmania braziliensis*.

Tropical kala azar is a visceral form of leishmaniasis occurring in India, China and the Sudan. Its causative organism is *Leishmania donovani*.

Fig. 2. Ringworm of the foot (*tupeks*). Grouped lesions on the sole with pustules, brownish scales, redness and induration.



tions whereas saprophytic organisms cause little inflammatory reaction. Severely inflammatory mycoses correspond to outspoken infectious allergy with a good tendency to heal spontaneously whereas lack of inflammation is paralleled by a deficient development of infectious allergy and a poor tendency to heal. Hematogenous dissemination of fungous elements especially in the inflammatory forms produces widespread eruptions of short durations the so called dermatophytids or mykids. 345'

Dermatomycosis

Ringworm of feet and hands (tinea pedis et manuum) (Figs 22 to 25) Tinea pedis develops between the toes on the soles and on the lateral parts of the feet. It originates as individual vesicles which later develop into lesions composed of vesico-pustules, crusts and brownish scales with underlying induration and surrounding erythema. In most instances the disease progresses slowly occasionally after marching especially in high boots and in hot weather a violent reaction may occur with the formation of numerous large blisters and pustules accompanied by lymphangitis and inguinal lymphadenitis. Mycelia can be demonstrated in the scales and in the coverings of the pustules. The most frequent causative organism is *Trichophyton interdigitale*. It may also be due to *Trichophyton rubrum*, *Epidermophyton floccosum* and other fungi. Sometimes a dermatophytid of the hands develops with formation of vesicles between the fingers and on the palms but often only lamellar scaling is visible.

Tinea manuum direct infection of the hands with fungi is less frequent than dermatophytosis of the feet. Scales, vesicles and pustules are found on the palms and between the fingers. Only the demonstration of fungi permits the distinction between a dermatophytid and dermatophytosis of the hands.

Tinea pedis is especially frequent during the summer months. Exposure to the infection occurs in swimming pools, on beaches, in gymnasiums and bath rooms. Some authors believe shoes are a significant source of reinfection but the value of disinfecting them with formaldehyde is disputed. Sweating and maceration of the feet should be reduced, frequent change of socks is recommended and waterproof boots should not be worn. Anticholinergic drugs such as Prantal, Banthine and Neo Banthine reduce sweating only in some cases.

Ringworm of the groin (tinea cruris et emarginatum) (Figs 26 and 27) Circular lesions develop in the inguinal and gluteal regions, they heal centrally and progress peripherally. On the spreading margin one sees vesicles, pustules, crusts and scales in which mycelia may be found. Similar lesions may occur in the axillae. The causative organism is often *E. floccosum* (*Epidermophyton inguinale*) but *T. interdigitale* may also be found. Not infrequently the same patient presents dermatophytosis of feet, hands, groins, axillae and gluteal region.



Fig 25 De matophyt d Erupt on of clear p nhead s zed ves cles on fingers



↑ Fig 23 Ringworm of the foot (*tinea pedis*)
Disseminated lesions with scaling and redness



Fig 24 Ringworm of the hand (*tinea manuum*)
Circumscribed erythematous and scaly lesions of the webs and dorsa of fingers



Fig 27 Ringworm of the buttock (*trinea cruris*: *eczema marginatum*). Circumscribed lesions of the buttocks with peripheral redness, scaling and a few pustules; there is central healing.

Fig 26 Ringworm of the groins (*tinea cruris* ec *ema marginatum*) Circumscribed lesion of the gro ns with peripheral redness scal ng and pustules the center is healing



Ringworm of the glabrous skin (tinea corporis) (Figs 28 to 30) Ringworm of the glabrous skin presents a picture similar to ringworm of the hands and feet but with lesions more sharply defined, round or polycyclic. The inflammation is most pronounced on the margin where erythema, small vesicles and pustules, crusts and scales are present. Towards the center of the lesion erythema and scaling decrease and the skin may appear completely normal. Usually there are a number of lesions of varying sizes which extend centrifugally. In certain cases a particular rhythm of the immunologic processes leads to concentric rings of activity. The fungi are found in scales and in the tops of pustules (Fig 21) in the form of double contoured segmented mycelia. They may also be found in lanugo hair. The infection is transmitted from man to man or from animal (dog cat cattle horse) to man. Frequent causative fungi are Trichophyton gypseum (Trichophyton mentagrophytes) and T. rubrum.

Inflammatory ringworm of the scalp (tinea capitis kerion) (Fig 31a) and of the beard (tinea barbae sycosis) (Fig 31b) On the scalp in the bearded region of the male or on other parts of the body covered with hair the fungi penetrate into the hair follicle alongside and within the hair shaft, causing a pustular folliculitis resembling furunculosis. The lesions are arranged in groups in round and polycyclic patterns. In inflammatory ringworm massive granulomas develop around the involved follicles within a few weeks leading to mushroom like granulation tumors with pus exuding from numerous follicular orifices. The Roman physician Celsus compared this condition to a honeycomb, hence the term kerion of Celsus.

The inflammatory follicular ringworm of the bearded region of the male is
sus staphylogenes or
whereas in staphylo-

Noninflammatory forms of tinea of the scalp cause round slightly scaly and red lesions in which the hair is broken off or shed. Slight scaliness covers a mildly erythematous area. An old name for this form of ringworm is herpes tonsurans.

In tinea of the hairy regions the fungi are found most readily in the broken off hairs which protrude from a purulent folliculitis. The hairs are made translucent with 10 to 20 per cent potassium hydroxide solution a process which can be accelerated by warming the slide. Mycelia and spores are found either in the hair itself (so-called endothrix form) or arranged around the hair (so-called ectothrix form). The less virulent fungi which are pathogenic for man only, are mostly endothrix. The more virulent strains which are also pathogenic for animals, are ectothrix. Ectothrix are common.

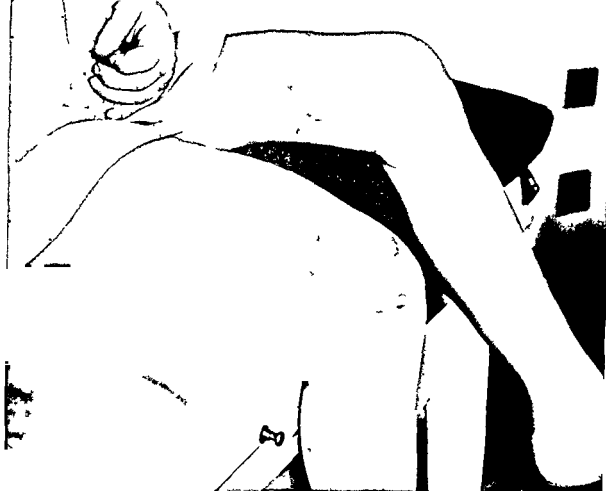
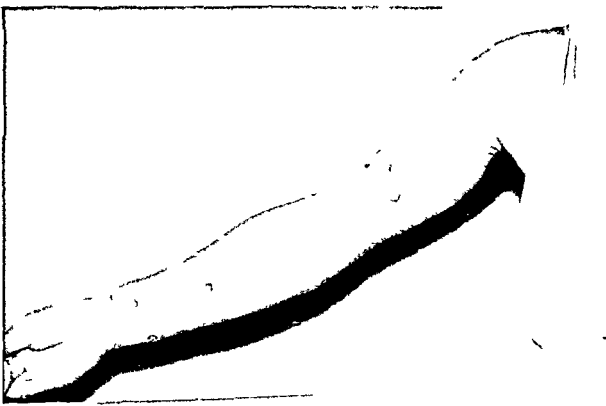


Fig 28 *Ringworm of the glabrous skin (trichophytosis)* Round circumscribed lesions with redness and scaling. The succession of central clearing and appearance of new lesions in the center produces concentric rings.

Fig 29 *Ringworm of the glabrous skin (trichophytosis)* Round lesions with redness and scaling. The larger plaques show central healing.



and s at



The infection often occurs from animal to man, farmers are frequently afflicted

The inflammatory trichophytoses have a tendency to clear up spontaneously, even extensive cases of kerion may heal with relatively little scarring and with regrowth of hair.

Trichophytids are not as common as dermatophytids, but occur especially in cases with numerous kerion lesions. They consist of lichenoid papules arranged around follicles and are found on the trunk and extensor surfaces of the extremities, the eruption is called lichen trichophyticus ✓

Noninflammatory ringworm of the scalp (tinea capitis) (Fig 32) Tinea capitis usually occurs in children of school age. On the scalp round lesions appear where fragile hairs are broken off a few millimeters above the surface. The sharply circumscribed lesions are covered with fine, powdery scales but signs of a more pronounced inflammation are lacking. On the glabrous skin there are only scaly, round, circumscribed plaques. Under the microscope the broken off hairs resemble a stick rolled in sand, for the shaft is surrounded by numerous spores. Bright greenish fluorescence of the infected hair under Wood's light is characteristic of tinea capitis due to microsporum. The causative organisms are chiefly *Microsporum audouinii* and *Microsporum canis*. Clinically the lesions caused by the two fungi may be indistinguishable, but *M. canis* can produce more inflammation. Occasionally there is severe inflammation, even the picture of a kerion, but this is unusual. Tinea capitis due to *M. audouinii* is a clinical example of noninflammatory ringworm. Tinea capitis is very contagious and often occurs as minor epidemics in schools, homes for children and orphanages. There is a tendency to spontaneous cure after puberty and it is rarely found among adults.

Treatment X-ray epilation is the best and quickest method of treatment. After the hair has fallen out an intensive antimycotic treatment should be started (see page 39). Where X-ray epilation is not possible or desirable, antimycotic ointments such as Salundek may be tried. They must be used for many months. If Asterol is used it should be applied to small areas only because of its neurotoxicity. Epilation can also be achieved by thallium acetate, the dose is 7 mg per kg of body weight. As the maximal dose is 240 mg per child, only children up to a weight of 34 kg can be treated by this method. Thallium is a strong poison and should be used with utmost caution.

Favus Originating from a follicular pustule a massive fungus culture in the form of a yellow, saucer shaped scutulum develops underneath the horny layer. Eventually this leads to atrophy of the underlying epidermis and follicles, causing permanent alopecia with scarring. The causative organism is *Achorion schoenleini* (*T. schoenleini*). This affection is very rare in the United States and in central Europe but still occurs epidemically in the Balkan countries. A rare

variety is the pityriasisforme favus without scutula, which resembles trichophytosis or seborrheic dermatitis. Treatment is the same as for resistant tinea capitis.

Ringworm of the nails (tinea unguium, onychomycosis) Ringworm may also involve the nails of the fingers and toes. The fungi penetrate into the nail plate from the distal or lateral borders and transform the nails into a crumbly mass. Ringworm of the nails is very resistant to treatment. The infected parts of the nails are removed with an electric drill or by other mechanical means to allow penetration of strong antimycotic solutions or ointments (page 39). Surgical removal helps only occasionally.

General Principles of Topical Treatment of Ringworm

Cases of ringworm with acute inflammation are treated like an acute dermatitis with soothing, mildly antiseptic medications (see pages 33 to 34). This applies to acute ringworm of the feet as well as of other areas. Fungicidal or peeling preparations are recommended after the acute state has subsided and for the so-called noninflammatory forms.

In mild cases of dermatophytosis powders and ointments may suffice which contain undecylenic acid (Desenex, Timofax, Tineafax), or Vioform, or 1 per cent Rivanol* in yellow petrolatum. Aqueous gentian violet solution, 1 per cent, often is useful even in the acute phase of tinea pedis or cruris.

More potent medications for dermatophytosis and ringworm of the glabrous skin are the following:

Sterosan ointment and paste (in Britain, Steroxin). Asterol ointment, or the following prescriptions:

Rx	Vioform	20 to 30
	Salicylic acid	24
	Yellow petrolatum <i>q s ad</i>	600
Rx	Precipitated sulfur	24
	Salicylic acid	24
	Yellow petrolatum <i>q s ad</i>	600

Arning's tincture is very helpful in persistent cases of ringworm. The formula is		
	Anthrarobin*	12
	Ichthyol	30
	Glycerin	60
	Ether and spiritus rectificatus, 70 per cent. ea <i>q s</i>	600

It may be fortified by the addition of tincture of iodine, as follows: tincture of iodine (10 per cent), 90, and Arning's tincture, *q s ad* 600.

* Rivanol and anthrarobin are available from Clinic Pharmacy, Marshfield, Wisconsin.

Fig 311 *Inflammatory trichoplytosis of hairy skin* Inflammatory tumor with redness, scaling and pustules (kerion)



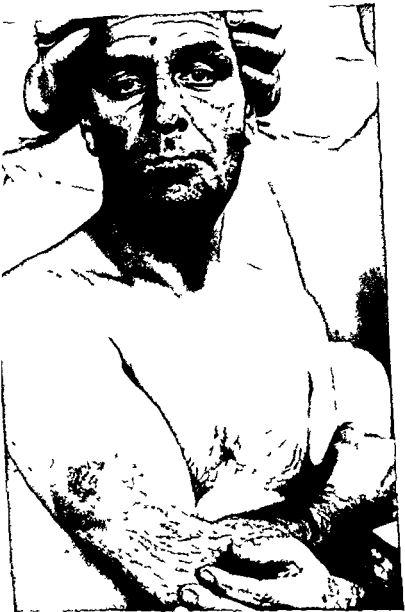


Fig 31b *Inflammation of beard (syphilis)* Circumscbed lesion of chin with redness swelling and numerous follicular pustules Strongly positive trichophyton reaction on the right arm is shown by redness swelling and pustules surrounded by a red halo

These alcoholic solutions are dabbed on twice a day and may be combined with any of the above mentioned ointments. In very chronic cases 1 to 2 per cent chrysarobin in Vaseline, Whitfield's ointment, or Wilkinson's ointment (compound sulfur ointment, N F) are helpful. Thorough peeling can be achieved with 10 to 25 per cent salicylic acid in Vaseline.

Erythrasma

In erythrasma brownish, well demarcated patches are found symmetrically in the groins or axillae. They show fine scaling when scraped. When the scales are examined with the oil immersion lens clusters of spores and short mycelia are found. The causative organism is *Microsporum minutissimum*, which cannot be cultured. The disease rarely produces subjective symptoms.

Treatment is the same as in pityriasis versicolor. If more resistant, Arning's tincture (see page 39) may help.

Pityriasis Versicolor (Tinea Versicolor) (Figs 33a and b)

This condition is characterized by round and polycyclic brown or yellow patches which occur singly or in large numbers on the trunk, less frequently on the extremities. Scales can be easily removed by scraping and show under the microscope groups of spores and short mycelia. The disease does not itch and usually does not bother the patient, except for cosmetic reasons. Often it is detected accidentally. The causative organism is *Microsporum furfur* (*Malassezia furfur*) which cannot be cultured. A special form of this condition is pityriasis versicolor alba, wherein the mycotic patches appear white in contrast to the pigmented surrounding skin. The ultraviolet rays of the sun are filtered out by the fungus and by the thickened horny layer which results from the infection. Therefore, the sunlight tans only the noninvolved skin and the mycotic skin appears relatively light. In pityriasis versicolor alba scales can be scraped off and fungi can be demonstrated, this permits differentiation from vitiligo and other forms of depigmentation.

Treatment. Mildly peeling lotions suffice for example

Rx	Salicylic acid	
	Resorcinol, aa	18
	Glycerin	48
	Spiritus, 70 per cent q s)	600
Rx	Saturated borax solution	
Rx	Undecylenic acid ointment compound (N F) (In Great Britain ung zinc undecen, B N F)	

These preparations are applied twice daily. As it is difficult to eliminate permanently the saprophytic fungus, the medication should be applied once a week even after the lesions have disappeared, in order to prevent a recurrence.

Fig 32 Ringworm of the scalp
(*tinea capitis*) due to *Microsporum*
audouinii. Round lesions covered by
gray scales. A few whitish broken
off hairs are present.

Fig 33a *Pityriasis versicolor*
Circumscribed slightly scaling le-
sions of a brownish or pinkish color





Moniliasis

There are numerous varieties of the *Candida* (*Monilia*) species they are yeastlike fungi found on and in the human body. They may become pathogenic under certain circumstances either because of increased virulence of the fungi or because of diminished resistance of the host as in diabetic or cachectic patients or following treatment with the so called broad spectrum antibiotics (see page 58) Local maceration of the skin also favors the development of monilial infections

Moniliasis occurring on the mucous membranes of the mouth is called thrush, round whitish fungus colonies occur in infants with nutritional disturbances and in the severely ill Moniliasis occasionally involves the lungs

Interdigital moniliasis also called *erosio interdigitalis oidiomycetica* or *blastomycetica* occurs following maceration of the skin of the webs of the fingers especially in housewives laundry women and bartenders It consists of a circumscribed erosion between the fingers with a red base and a whitish macerated edge Spores and mycelia of *C. albicans* are found in the horny layer of the edge of the lesion

A similar picture is seen in intertrigo of the skin folds of fat people beneath the breasts on the abdomen and in the groins around the eroded parts crops of pustules can be seen *Balanitis vulvitis* and *vaginitis* may also be caused by *Candida*. The most common causative organism is *C. albicans*

Treatment Superficial moniliasis may respond to wet compresses application of borax glycerine or a borax shake mixture as follows

Rx	Borax	60
	Zinc oxide talc aa	100
	Glycerin	
	Distilled water aa q s	600

Aqueous gentian violet solution 1 per cent is very effective but must be used

by weaker stronger antimycotic lotions or ointments

by vit to be helpful in the latter condition When internal organs are involved Mycostatin should be tried internally

Fig 33b *Pityriasis versicolor alba* Numerous white spots coalescent on the center of the back. Note contrast with the suntanned normal skin.



Fig. 34 North American blastomycosis (Courtesy of Dr. Louis Brunsting, Mayo Clinic, Rochester, Minnesota)

Deep Mycoses

Blastomycosis

North American blastomycosis (✓ Gilchrist's disease) This disease occurs in the United States and Canada. It begins as a papule which breaks down and spreads slowly by peripheral extension. Round and polycyclic lesions are formed, their elevated and verrucous borders having a characteristic appearance (Fig 34). It is assumed, although not proven, that the skin lesions are often primary. They are usually found on the exposed parts of the body, especially the face. There is also a systemic form of blastomycosis, the infection usually enters through the respiratory tract. Lungs and bones, but also liver, spleen, kidneys and brain may be involved. Even in these cases, often the first symptom noticed by the patient is an ulcer of the skin or a subcutaneous nodule or abscess.

The disease is caused by *Blastomyces dermatitidis*. A definite diagnosis is made by the microscopic demonstration of blastomyces or by culture. A positive blastomycin test is suggestive. Histologically there is abscess formation and chronic inflammation, with giant cells and necrosis. The prognosis in cutaneous blastomycosis is favorable, it rarely causes death. However, systemic blastomycosis usually is fatal.

Treatment Cutaneous blastomycosis may respond to iodine or x-ray therapy, aided by desensitization and immunization with vaccines. Stilbamidine therapy has been effective in disseminated blastomycosis. Apparently the new anti-fungal antibiotic, Amphotericin B, is most promising.

South American blastomycosis This disease is caused by *Blastomyces brasiliensis*. In disseminated cases there may be numerous granulomatous and ulcerated lesions of the skin and mucous membranes of the mouth, the internal organs often are involved. Lymph node enlargement is a characteristic finding.

European blastomycosis (cryptococcosis torulosis) This condition is caused by *Cryptococcus neoformans*. It primarily involves the nervous system, cutaneous lesions are uncommon.

Histoplasmosis and coccidioidomycosis Other deep mycoses which only rarely cause primary lesions of the skin are histoplasmosis caused by *Histoplasma capsulatum* and coccidioidomycosis, caused by *Coccidioides immitis*. However, secondary involvement of the skin is not uncommon in the latter.

Sporotrichosis

Sporotrichosis most frequently appears as a subcutaneous nodule, the sporotrichotic chancre which may become necrotic and ulcerated. Secondary subcutaneous nodules develop along the lymphatics (Figs 35a and b). The appearance of a primary lesion with its satellites gives sporotrichosis a typical



Fig. 35b *Sporotrichosis* (same patient as Fig. 35a) with nodular lymphangitis on the upper arm (Courtesy of Dr. Harold Perry, Mayo Clinic, Rochester, Minnesota)



Fig 35a *Sporotrichosis* Sporotrichotic chancre on the dorsum of the hand with satellite lesions on the forearm (Courtesy of Dr Harold Perry, Mayo Clinic Rochester, Minnesota)

clinical picture easy to recognize The disease occurs most often on hands, fingers, arms and legs, but sometimes on the face There are rare generalized cases of sporotrichosis occasionally involving the viscera

Sporotrichosis is caused by *Sporotrichum schenckii*, the infection often is contracted from plants following slight injury of the skin The diagnosis is confirmed by culture of the causative organism

In the United States sporotrichosis occurs most frequently in the Midwest around the Mississippi Valley With adequate treatment the prognosis is usually excellent

Treatment Potassium iodide is practically a specific drug for sporotrichosis Ten drops of a saturated solution in water or milk are given three times daily initially The dose is increased by 3 to 5 drops three times a day, up to 30 to 40 drops three times a day, unless intolerance develops Treatment should be continued for at least a month after clinical cure has been achieved

Actinomycosis

Actinomycosis is characterized by subcutaneous nodules and boardlike infiltrations which tend to form fistulae The pus contains granules which can be seen with the naked eye Microscopically one recognizes the "ray fungus", a fungous mass with radiating mycelia on the periphery The fungi may be stained with methylene blue they are Gram positive The most common loca-



Fig 36a *Staphylococcic impetigo* with flat crusts and a circinate lesion on of the nose (From Epstein S *Arch Dermat & Syph* 42 840 1940)

Ecthyma (Fig 37b)

Ecthyma is usually seen on the lower legs rarely on other parts of the body it consists of round and oval purulent ulcerations about 1 to 2 cm in diameter surrounded by erythema and induration The affection often lasts for weeks or months and leaves scars The causative organism is usually a streptococcus only occasionally a staphylococcus

Other Forms of Nonfollicular Pyodermas

The chancre like pyoderma of the face consisting of an erosion with marked induration of the surrounding skin is another form of nonfollicular pyoderma

bovis

Treatment Parenteral penicillin is given daily 600 000 units of procaine penicillin up to 12 million units or Aureomycin Terramycin or sulfonamides are used internally These methods have superseded the older treatment with potassium iodine and/or x ray irradiation

Pyodermas

The term pyoderma covers skin diseases which are caused by pyogenic bacteria such as staphylococci and streptococci These cocci are obligate contaminants of the human skin Through a decrease of general or local resistance of the host or following small external traumas they may multiply and increase in virulence These cocci with increased virulence become infectious for the host and other individuals

Nonfollicular Pyodermas

The nonfollicular pyodermas are not connected with the adnexa of the skin

Impetigo Contagiosa (Figs 36 and 37)

The primary lesion of streptococcic impetigo is a subcorneal vesicle or pustule Where the skin is covered with a relatively thin horny layer the blister quickly bursts and an erosion becomes covered with a crust or scale Clinically there are two forms of impetigo In one there are flat brownish crusts or flaccid bullae especially on the face (Fig 36a) and the lesions are circinate somewhat resembling ringworm (Fig 36b) This form of impetigo is caused by staphylococci In warm climates staphylococcic impetigo may cause large blisters on the body Pemphigus neonatorum is an older term for staphylococcic impetigo of the newborn It is a very contagious pyoderma and may extend all over the body in the form of an exfoliative dermatitis known as Ritter's disease The other form of impetigo is characterized by golden yellow crusts which are spread in coin sized patches over the face and hands (Fig 37) When the crusts are removed a red easily bleeding and oozing surface is exposed This form of impetigo is caused by streptococci Where the horny layer is thicker as on palms soles and fingers blisters and pustules persist longer

Impetigo is chiefly a disease of children and usually heals without scars The staphylococcic form is especially contagious whole families may become infected Streptococcic impetigo often occurs as a complication of scabies or eczemas



Fig. 37a *Streptococcic impetigo contagiosa*
showing heavy yellowish crusts





Fig 36b *Staphylococcic circinate impetigo* resembling ring worm (From Epstein *S Arch Dermat & Syph* 42 840 1940)

it has also been called impetigo vegetans. In angular stomatitis or perleche there are linear erosions and fissures of the corners of the mouth with redness of the surrounding skin. This condition may have several causes. Increased salivation, malocclusion of the mouth from ill fitting dentures, vitamin or iron deficiencies and secondary infection with streptococci or *C. albicans* must be considered. Ulcus tropicum is a chronic ulcer found mostly on the lower legs. It is secondarily invaded by fusiform rods and spirochetes (Plaut Vincent's symbiosis).

Follicular Pyodermas

The follicular pyodermas are connected with the hair follicles or sweat glands. They are caused by staphylococci (*Micrococcus pyogenes*).

Folliculitis

When staphylococci penetrate the hair follicle a yellow pustule develops in the orifice of the follicle, often with a hair visible in the center. This folliculitis occurs usually in groups on the face, neck or trunk.



Fig 40 *Furunculosis* A group of infected follicles with abscess formation (furuncles)



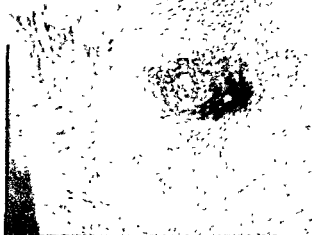


Fig 38 *Furuncle of the nape of the neck.*

Carbuncle of the chin



Furuncle (Fig 38)

In case of marked virulence of the staphylococcus or of poor resistance of the host, the folliculitis turns into a furuncle. An abscess is formed around the hair follicle which is destroyed and expelled as a necrotic plug. The inflammation around the follicle is intensive and is accompanied by redness, marked induration and pain. Common locations of furuncles are the back of the neck, the face, the upper lip and the genital area. Furuncles of the upper lip and nose are dreaded because of possible extension to the sinus cavernosus.

Carbuncle (Fig 39)

If a group of adjacent follicles becomes infected or if the subcutaneous tissue is invaded, the resulting lesion is referred to as a carbuncle. The necrotic follicles may turn into a subcutaneous abscess. Yellow pus can be expressed from the several openings.

Furunculosis (Fig 40)

If the patient suffers from recurrent furuncles, one refers to this condition as furunculosis. The main cause is the inability of the body to produce a satisfactory immunity to the staphylococci of the skin.

Sycosis Vulgaris (Sycosis Simplex)

When groups of follicular pustules occur in the bearded region, the condition is called sycosis vulgaris (staphylococcic sycosis). This is usually chronic and recurring because it is difficult to eliminate the staphylococci and to improve the lowered resistance of the patient.

Apocrine Sweat Gland Abscesses (Hidradenitis Suppurativa) (Fig 41)

Groups of hazelnut sized cutaneous nodes develop in the axillae, often adherent to the inflamed overlying skin. Gradually some of them develop into abscesses and discharge their purulent contents. Closely adjacent nodules may coalesce and form a single indurated tumor. Hidradenitis suppurativa is an infection of the apocrine sweat glands caused by staphylococci, often it is chronic and recurrent.

There are other more rare forms of follicular pyodermas. Dermatitis papillaris nuchae is a chronic disease consisting of groups of follicular pustules of the back of the neck. Keloidal scars transform this area into a boardlike plaque, often with papillomatous hypertrophy. In pyoderma suffodiens et abscedens capitis (perfolliculitis capitis abscedens et suffodiens) subcutaneous abscesses and fistulas occur on the scalp.

Fig 42 Popular diaper rash (*pseudosyphilide posterosive*) Eroded papules in the anogenital region of an infant



Furuncle (Fig 38)

In case of marked infection of the hair follicle, the host the follicle which is destroyed and the surrounding tissue is inflamed. Induration and pain. Common locations are the face, the upper lip and the back. They are dreaded because of possible extension to the brain.

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Fig 42 Papular diaper rash (*pseudosyphilis posterio-
sive*) Eroded papules in the anogenital region of an infant



Furuncle (Fig 38)

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When groups of follicular pustules occur in the bearded region, the condition is called sycosis vulgaris (staphylococcic sycosis). This is usually chronic and recurring because it is difficult to eliminate the staphylococci and to improve the lowered resistance of the patient.

Apocrine Sweat Gland Abscesses (Hidradenitis Suppurativa) (Fig 41)

Groups of hazelnut sized cutaneous nodes develop in the axillae, often adherent to the inflamed overlying skin. Gradually some of them develop into abscesses and discharge their purulent contents. Closely adjacent nodules may coalesce and form a single indurated tumor. Hidradenitis suppurativa is an infection of the apocrine sweat glands caused by staphylococci, often it is chronic and recurrent.

There are other more rare forms of follicular pyodermas. Dermatitis papillaris nuchae is a chronic disease consisting of groups of follicular pustules of the back of the neck. Keloidal scars transform this area into a boardlike plaque, often with papillomatous hypertrophy. In pyoderma suffodiens et abscedens capillitii (perifolliculitis capitis abscedens et suffodiens) subcutaneous abscesses and fistulas occur on the scalp.

Treatment of Pyodermas

Antibiotics and Other Antibacterial Agents

Systemic treatment Penicillin is still the most active antibiotic against infections with Gram positive bacteria, except for penicillin-resistant strains of *Micrococcus pyogenes* (*Staphylococcus pyogenes*). It should not be used in patients known or suspected to be sensitive to penicillin and it is better avoided in cases complicated by dermatophytosis of the feet. Erythromycin is also active mostly against Gram positive bacteria. The so-called broad spectrum antibiotics, the tetracyclines, are effective against Gram positive as well as Gram negative organisms.

The dosage for adults and mode of administration are as follows

Penicillin In almost all but the most severe skin infections oral penicillin suffices, 250 mg (400,000 units) of penicillin V or other penicillin preparations are given three or four times a day. In the case of intramuscular injections 600 000 units daily is an adequate dose for most skin infections.

Erythromycin (Ilotycin, Erythrocin) This is started at a dose of 250 mg four times a day and is later reduced to 100 mg four times a day.

Broad spectrum antibiotics These are tetracycline (Achromycin, Panmycin, Polycycline, Tetracycl), chlortetracycline (Aureomycin), oxytetracycline (Terramycin), and chloramphenicol (Chloromycetin). The usual dose is 250 mg four times a day. Prolonged use of the broad spectrum antibiotics may cause pruritus and irritation of the anogenital region followed by *Monilia* invasion. This can usually be prevented by the simultaneous use of large amounts of vitamin B complex plus vitamin C, or by using the so-called stress fortified antibiotics which provide these vitamins for example, Achromycin S-F, Terramycin S-F or Tetracycl S-F.

Sulfonamides Most frequently used now are (1) mixtures of several sulfonamides containing equal parts of sulfadiazine, sulfathiazine and sulfamerazine (Triple Sulfa, Trisulphonamide tablets, B N F) or of sulfacetamide, sulfadiazine and sulfamerazine (Tricombisul), (2) the newer Gantrisin (sulfisoxazole), or (3) Elkosin (sulfisomidine). Treatment is started with 2 gm as the initial dose and continued with 1 gm four times a day. Severe infections require larger amounts, for prolonged treatment in recurrent pyodermas much smaller maintenance doses are used, sometimes as little as 0.5 gm once or twice daily.

Acute skin infections often require administration of antibiotics or sulfonamides for 5 days, in other instances longer treatment is advisable.

Whenever a skin infection does not respond satisfactorily to an antibiotic or sulfonamide, sensitivity studies of the causative organism are indicated.

Topical treatment Bacitracin and neomycin are preferred for local application because they are rarely employed internally, however, erythromycin

(Stotycin) Aureomycin, and Terramycin ointments also may be used, since these antibiotics rarely sensitize. Penicillin or sulfonamides should not be applied topically.

Special Considerations in the Treatment of Individual Pyodermas

In extensive impetigo and ecthyma systemic penicillin is indicated. In staphylococcal impetigo one may prefer erythromycin or a broad spectrum antibiotic. If there are only a few lesions local treatment with antiseptic ointments or lotions suffices. Topical antibiotics are very effective. Between applications of the ointment the skin is cleansed with wet compresses of a mild antiseptic, aluminum subacetate or Alibour's lotion (see page 133). In pemphigus neonatorum systemic penicillin is used. The blisters are opened and treated with 3 per cent silver nitrate solution and a 1 per cent gentian violet solution is applied, but no baths are given and no ointments are applied. This dry regime is continued until the infection has stopped spreading at which time antibiotic ointments may be used.

In follicular pyoderma parenteral or oral penicillin may be tried but as there are many penicillin resistant staphylococci nowadays other antibiotics often are more effective. Systemic antibiotics are usually reserved for more severe cases. Sulfonamides may also be used. Local treatment of folliculitis

with antibiotic ointment as mentioned above. To disinfect the whole skin potassium permanganate baths are indicated. For an adult the quantity is 10 g for a tub. Since the advent of sulfonamides and antibiotics surgical treatment of furuncles, carbuncles or sweat gland abscesses is rarely necessary, usually one can wait for spontaneous perforation and for the expulsion of the pus and the follicular plug. In sycosis vulgaris antibiotic ointments are helpful when combined with wet compresses (see above). After the pyoderma has healed regular disinfection of the skin is indicated with 1 per cent Zephiran or Alibour's lotion or an alcoholic solution containing 2 per cent salicylic acid.

In furunculosis underlying conditions such as diabetes, anemia or focal infection should be taken care of. One tries to increase the general resistance of the patient with rest, vitamins, etc. and to build up the specific immunity with either commercial staphylococcal vaccines plus staphylococcal toxoid or with autogenous vaccines. In resistant cases of sycosis simplex desensitization with diluted staphylococcal vaccine (1:10,000) or better with a combination of staphylococcal and streptococcal vaccines with trichophytin and oidiomycin given intradermally for a long period of time, is helpful.

Intertrigo (Fig 42)

"Intertrigo" is a term for sharply outlined erythematous patches in skin folds, such as occur in the groins and in the anal, abdominal and intramammary areas, especially in fat people. Intertrigo may be caused by various microbes such as *C. albicans*, by cocci and by other saprophytes, diabetes mellitus may be an underlying factor.

A special form of intertrigo in infants is the erythema papulosum posterosivum, a papular form of diaper rash probably due to maceration of the skin from urine and secondary infection. The superficially eroded lesions (Fig 42) resemble papular syphilis, hence the name "pseudosyphilide posterosive".

Treatment: Antiseptic powders are helpful, among them are Vioform, Sterosan, Desenex, or some combination, such as Pulvogen, containing hexa chlorophene, ichthammol and zinc oxide in an absorbent base. These powders are used alone or alternatively with hexachlorophene shake lotions or pastes (pages 133 to 134). Cleansing is carried out with wet compresses of aluminum subacetate or potassium permanganate solution 1:6000. In resistant cases the additional application of a 1 per cent aqueous gentian violet solution is very helpful, as is vioform or vioform hydrocortisone lotion.

Erysipelas ("St. Anthony's Fire")

Erysipelas is characterized by a sharply demarcated acute redness and swelling of the skin which slowly progresses, it is usually accompanied by chills, fever and feelings of malaise. In severe cases blisters may be formed or even necrosis may follow, with poor resistance erysipelas migrans may occur. Following repeated attacks of erysipelas elephantiasis may develop because the lymphatic vessels and lymph nodes may be destroyed by the infection.

The causative organism is *Streptococcus erysipelatis*, a β hemolytic streptococcus. The incubation period is 1 to 2 days. Erysipelas occurs more often in patients with chronic skin diseases such as lupus vulgaris, leg ulcers, fissures and surgical wounds. The formerly dreaded disease now can be well controlled by chemotherapy.

Treatment: Penicillin, sulfanilamide or sulfadiazine or broad spectrum antibiotics are used (see pages 58 to 59). Local treatment has become unnecessary for the acute condition, but recurrent erysipelas of the face may be prevented by the continuous use of an antibiotic ointment at the portal of entry of the infection, for example the nostrils or external ear.

Erysipeloid (Swine Erysipelas)

Erysipeloid shows circumscribed redness of the skin, similar to erysipelas but without marked general symptoms. It is usually seen on the fingers and hands.

sometimes with a local arthritis. Spontaneous cure may occur within a few days.

The causative organism, *Erysipelothrix rhusiopathiae*, is transmitted to man from the meat of infected animals, for example, pork, fish and game. It may also be transmitted by bites of insects or ticks. Erythema chronicum migrans perhaps is a variety of erysipeloid. The treatment is the same as that for erysipelas.

Granuloma Pyogenicum

The lesion consists of a spherical, red tumor from pea to cherry size, its surface often is covered by a crust. When the crust is removed one sees an easily bleeding erosion. Histologically the condition consists of a highly vascular granulation tissue. It is probably an infectious process. At times it may be difficult to distinguish it from an amelanotic melanoma.

Treatment. Some cases respond to an antibiotic ointment, usually electro-surgical or surgical removal is necessary.

Other Bacterial Diseases of the Skin

Anthrax

Another rare bacterial infection of the skin is anthrax. This is a hemorrhagic, necrotizing, furuncle-like lesion which may lead to septicemia. The causative organism is the Gram positive *Bacillus anthracis*. The infection often is transmitted from animals.

Treatment. Parenteral or oral treatment with penicillin, Aureomycin, Terramycin, Chloromycetin or streptomycin is effective.

Rhinoscleroma

Rhinoscleroma is characterized by a hard, nodular, nonulcerating, thickening of the upper lip, nostrils or throat. The causative organism is a Gram negative bacillus. Rhinoscleroma is resistant to therapy but streptomycin and *p*-aminosalicylic acid may be tried.

Tularemia

Tularemia is an epidemic disease of rodents which may be transmitted to man. The disease causes ulcerations of the skin, lymphadenitis, fever and sometimes septicemia. The disease is caused by *Pasteurella tularensis*.

Treatment. Terramycin, Chloromycetin, Aureomycin or streptomycin are recommended.

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Skin Diseases Caused by Animal Parasites

Skin Diseases Caused by Mites

Scabies (the "Itch") (Figs 43 to 45)

Human scabies is caused by a mite, *Sarcoptes scabiei*. The fecund females dig burrows in the epidermis, where they deposit their ova and feces. The burrows are often slightly elevated and are especially prominent in areas with a thick horny layer, for example the fingers, palms and soles. By a tangential excision of these burrows the mites, ova and feces can be demonstrated microscopically. Special locations of scabies are the hands, feet, anterior axillary folds, elbow and penis, but the eruption may cover the whole body with the exception of the head. Patients complain especially of nocturnal pruritus because the parasites are more active in the warmer temperature of the bed. Scabies is transmitted from one individual to another, often as a result of sleeping in the same bed. Small epidemics are observed in families, homes for children, or army barracks. A special rare form is Norwegian scabies characterized by psoriasis like lesions or by an erythroderma the scales of which are loaded with mites. Lack of host resistance may be a factor. The diagnosis often is made only after other persons, such as nurses, have become infected. These secondary cases present the picture of ordinary scabies.

Treatment On two consecutive days an ointment containing hexachloro cyclohexane (for example Kwell or Gammexane) or crotamiton (Eurax) is applied to the whole of the skin surface below the neck. Children should receive a second course of treatment after 2 weeks. On the third day the patient takes a bath and changes underwear and sheets. It is also necessary to treat contacts such as sexual partners and other members of the family. Where the diagnosis is in doubt a trial with antiscabietic treatment often is helpful (diagnosis *ex juvantibus*).

Animal Scabies

ma
are found on the skin of animals, especially from its bedding. Scabies of pigeons and chickens (*Dermanyssus avium*) originates from infected dovecotes or chicken coops. Horse scabies (acarosis equi) is observed in grooms. *Acarus canis* and *acarus cati* cause eruptions in persons who handle dogs and cats. Treatment of the infected animals and disinfection of their living quarters are the chief means of their control.

Two other mites are encountered less often. *Pediculoides ventricosus* lives on the eggs and larvae of grain and corn moths, which grow in grain and other

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Animal Scabies

The mites causing animal scabies may produce a papular itching eruption in man through contact with infected animals or with their bedding. They rarely are found on the afflicted humans but may be recovered from the animal and especially from its bedding. Scabies of pigeons and chickens (*Dermanyssus avium*) originates from infected dovecotes or chicken coops. Horse scabies (*Acarus equi*) is observed in grooms. *Acarus canis* and *acarus cati* cause eruptions in persons who handle dogs and cats. Treatment of the infected animals and disinfection of their living quarters are the chief means of their control.

Two other mites are encountered less often. *Pediculoides ventricosus* lives on the eggs and larvae of grain and corn moths which grow in grain and other

legumes, it causes itching eruptions in grocers and grain handlers. The larvae of *Trombicula autumnalis* which exists on the foliage of beans and on bushes, grasses and flowers, causes a papular itching eruption in passers-by and in agricultural workers

Tick Bites (*Ixodes Ricinus*)

Wood ticks several millimeters long may be encountered in forests. The head of this mite penetrates into the skin and the tick sucks itself full of blood. Usually there is only a minor inflammation, but rarely probably when the ticks are infected, erysipelas like eruptions occur. Tick bites may progress as ring shaped red lesions (*erythema centrifugum migrans*) or they may lead to febrile virus diseases with meningitis. Ticks can be the vector of Rocky Mountain spotted fever, tularemia and tick paralysis.

Treatment The application of a drop of oil kills the tick, which falls off several hours later. Alternatively, the affected skin with the head embedded *in situ* may be cut off with scissors.

Skin Diseases Caused by Insects

Pediculosis (Figs 46 to 49)

Pediculosis corporis Body lice deposit their eggs in the folds of clothing. Their bites cause small papules, scratch marks, pigmentation, pyodermas and scars are also seen. The afflicted persons often are tramps and vagabonds hence the name 'vagabond's disease', (Fig 47)

Transmission occurs in primitive sleeping conditions. In war time whole bodies of troops and segments of the population may be infested. *Pediculosis corporis* is important for

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hexachlorocyclohexane powder (Gammetane), fumes, or DDT (Dicophane), constitutes the recommended treatment.

Pediculosis capitis The head louse (*Pediculus capitis*) lives in the hair of the scalp and fastens its ova (nits) to the base of the hair, especially in the region of the nape and behind the ears. Where suspicion of *pediculosis capitis* exists, one has to search this region for the firmly attached nits. The nitrification and the

Treatment A short haircut is the best treatment for boys, application of antiparasitic preparations such as Kwell or Topocide, kills the nits which afterwards must be softened with oil and removed with a fine comb.

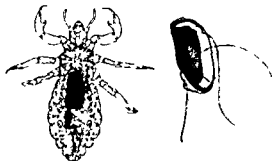


Fig 46a *Pediculus corporis* with ovum
(magnification $\times 15$)

Fig 46b *Pediculus capitis* with nit adherent
to the hair (magnification $\times 25$)

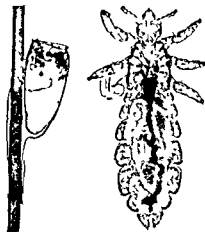
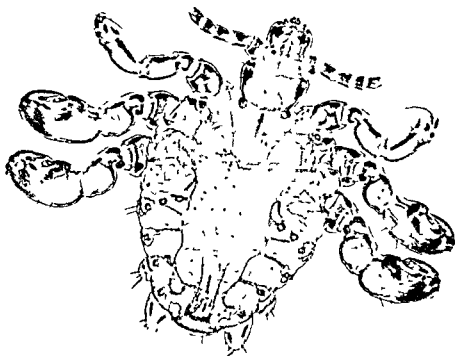


Fig 46c *Pediculus pubis* (crab louse) (ma-
gnification $\times 50$)



g 47 *Vagabond's skin* Papules excoriations scars hyperpigmentation
 mentation and folliculitis following persistent infestation with body lice



Pediculosis pubis The crab louse (*Phthirus pubis*, *Pediculus pubis*) lives in the hairy regions of the body and deposits its nits at the base of the hairs. Only exceptionally, in children, is the hair of the scalp involved. The preferred location is the pubic area but in extensive cases the crab louse can also be found in the axillae and even in the eyebrows. The bite may lead to a minor hemorrhage, causing bluish spots called "maculae caeruleae". In suspicious cases the body hair must be searched for lice and nits with a magnifying glass. Transmission occurs usually through sexual intercourse but may be due to the use of infested blankets or clothing, in which the lice can survive for several hours.

Treatment The same antiparasitic ointments or lotions are used as in pediculosis capitis.

Flea Bites

The human flea (*Pulex irritans*) often causes a small hemorrhage and in susceptible persons, a wheal or an itching papule. The bites of the human flea usually are arranged in groups on the covered parts of the body. Man is also attacked by cat and dog flea, mostly on the lower extremities and especially the calves.

Treatment When symptomatic treatment is required an alcoholic solution containing 2 per cent menthol suffices, or an antihistaminic ointment or hydrocortisone lotion. In case of severe infestation it is necessary to disinfect the apartment, especially the cracks in the floors and walls, where the eggs are deposited, disinfection may be carried out with the fumes of hexachlorocyclohexane or with DDT powder. In case of animal fleas cats and dogs have to be treated and rugs should also be disinfected.

Bedbug Bites

Bedbugs (*Cimex lectularius*) nest in beds, behind wallpaper and in the cracks of walls. They bite people, especially at night, while they are asleep. The bites may produce wheals, papules or blisters usually on those parts of the body which are not covered by the nightgown—on hands, feet, neck and face.

Treatment is the same as for fleas (see above).

Bites by Mosquitoes Bees, Wasps and Hornets

Mosquito bites are usually harmless, they may produce small papules and by secondary infection pustules and pyodermas. Stings from bees, hornets and wasps occasionally lead to severe general symptoms, even shock which require injections of calcium, antihistamines or adrenaline, or central nervous stimulants such as Coramine (Nikethamide). Prompt local relief is obtained by hydrocortisone lotions or ointments.



Fig 48 *Pediculosis vestimenti* Itching papules on the trunk following infestation with body lice

Fig 49 *Pediculosis pubis* Crab lice can be seen in the hairy region as brownish spots. The bluish spots (taches bleues) represent hemorrhagic reactions to the bite of the pubic lice

Skin Diseases Caused by Worms

Certain infestations with worms may cause skin lesions. Pinworms (*Oxyuris*) cause anogenital pruritus and occasionally general pruritus. In children they may produce a papular eruption of the buttocks and lower legs often mistaken for eczema. *Ascaris* sometimes sensitizes the skin of its host and sometimes causes urticaria, pruritus and bronchial asthma. Löffler's transitory eosinophilic infiltrate of the lungs may develop occasionally an eosinophilic epididymitis or prostatitis occurs. Tapeworms (*Taenia saginata*, *Taenia solium*) occasionally cause urticaria, pruritus and prurigo. The skin lesions disappear after successful treatment of the worms with antihelminthics.

Treatment Pinworm infestation responds to oral Terramycin or gentian violet. Roundworms as well as pinworms are treated effectively with piperazine hexahydrate (Syrup of Antepar).

A worm infestation which afflicts the skin only is swimmer's itch or schistosomiasis caused by *Cercaria ocellata* (Fig. 50). The normal hosts of these worms are waterfowl but the larvae develop in a snail *Lymnaea stagnalis* which exists in lakes. The worms in the cercaria stage attack persons who are bathing. They penetrate the skin causing an urticarial eruption which persists for several days. After a few days the cercaria die in the skin as humans are not a true host for this parasite. Swimmer's itch is observed on many lakes of the United States, Switzerland, Germany and New Zealand and a similar dermatitis is seen in workers in rice fields. The dermatitis disappears spontaneously. Control of the cercaria infested snails can be achieved with copper sulfate.

Creeping disease occurs in the tropics and also in the southern part of the United States but rarely in Europe. It is caused by a migrating larva which leaves a linear, bizarrely tortuous, papular eruption. The causative organisms are *Schistosoma mansoni* and *Ancylostoma braziliense*.

Another rare tropical disease, dracontiasis, is caused by the guinea worm. Subcutaneous nodules are found on the lower legs from which the worm protrudes spontaneously or after incision. The parasite may reach the length of 1 m.

Filaria caused by *Filaria bancrofti* is an infectious disease of the lymph nodes, destruction of which leads to elephantiasis of the scrotum and the lower extremities.

Virus Diseases of the Skin

Herpes Simplex (Fig. 51)

Herpes simplex consists of groups of pinhead sized vesicles and pustules on a reddened, sometimes cushion like swollen base. Occasionally larger blisters are seen and slightly painful swelling of the regional lymph nodes may occur. Herpes simplex heals within a few days, the blisters drying up to crusts.

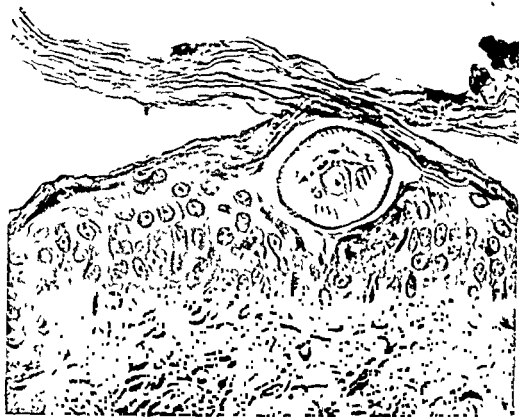


Fig 50 *Cercaria dermatitis* ("swimmer's itch") Histologic picture of experimental infection 5 hours after the penetration of the cercaria

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Fig 51 *Herpes simplex labialis*



removal of the crusts reveals numerous small erosions. Frequent locations are the lips (herpes labialis) and the genital regions (herpes progenitalis). On the mucous membrane of the mouth, the glans penis and the vulva, only the erosive stage is usually seen. The lesions may be accompanied by pruritus. Rare com-

easily be transmitted to the cornea of rabbits. In humans the eruption may be precipitated by minor lowering of resistance, as in menstruation, by febrile

cure secondary infection. Herpes of the cornea should be referred to an ophthalmologist. In recurrent herpes simplex, repeated vaccinations with small-pox vaccine at 2 week intervals may be tried, at times the herpes can be suppressed or at least the intervals between recurrences may be lengthened. Prolonged intranasal use of an ointment containing hydrocortisone and an antibiotic may be tried in refractory cases of herpes of the lips and nose.

Herpes Zoster ("Shingles") (Fig. 52)

Groups of pinhead sized vesicles surrounded by redness and swelling occur on one side of the body in the distribution of one or more posterior roots of cranial or spinal nerves. Within a few days they become pustules and then dry up, forming crusts which conceal small erosions. The lesions heal in 2 to 3 weeks. In severe cases there is a necrosis of the base of the pustules (herpes zoster necroticans) which heals with small scars. In exceptional cases there is more massive gangrenous destruction (zoster gangrenosus). The skin manifestations are often accompanied, or even preceded, by neuralgic pain which in 6 per cent of the cases continues for several weeks after the eruption has healed. In less than 1 per cent of the cases herpes zoster is followed by severe neuralgia which persists for years. In people over 40 years of age the course is usually more protracted. Very rarely, one finds herpes zoster duplex, a condition in which both sides of the body are involved simultaneously. Complications are herpes zoster generalisatus which is a generalized chickenpox-like eruption, serious meningitis, involvement of motor nerves with facial paresis, and transitory deafness (zoster oticus). Herpes zoster of the cornea occurs when the ophthalmic branch of the trigeminal nerve is involved, and vesicles appear on the mucous membrane of the mouth if the maxillary branch of this nerve is affected.

Histologically herpes simplex and herpes zoster produce a so called balloon inflammatory in

has been made probable by the demonstration of Paschen bodies in the lesions and by transmission to children. As in herpes simplex, various factors favor its onset, for example, infectious diseases, trauma, fatigue and internal arsenic medication. Herpes zoster leaves a permanent immunity

Treatment A specific treatment does not exist as yet. Pain is relieved by aspirin or, if more severe, by codeine or Demerol. In Britain Pethidine is preferred over Demerol. Local treatment consists of the application of antiseptic ointments or lotions (hexachlorophene, antibiotics, Vioform). Autohemotherapy seems to be helpful. Cortisone should be reserved for severe cases only. In case of secondary infection or necrosis internal antibiotics are indicated. Deep x ray therapy applied to the area of the involved ganglia often relieves the neuralgia. Zoster of the cornea requires treatment by the ophthalmologist.

Warts and Wartlike Tumors (Infectious Acanthopapillomas)

Verrucae Planae Juveniles (Juvenile Warts) (Fig 53)

Juvenile warts are small, flat, polygonal, skin colored or slightly brownish papules, sharply outlined and a few millimeters in diameter. Juvenile warts, often in enormous numbers, occur in groups on the uncovered parts of the body such as the face, hands and forearms. They are found especially in children and younger people up to 25 years of age. Treatment is described under "Verrucae Vulgares".

Verrucae Vulgares (Fig 54)

Warts are papular, hyperkeratotic tumors a few millimeters in diameter, with a rough, sometimes fissured, horny surface, they occur singly or in groups especially on areas of the body not covered by clothing but also on the soles and scalp. Special forms develop around the fingernails as hyperkeratotic, plaque-like tumors with deep fissures. On the soles they form painful indurations beneath the thick horny layer (plantar warts), on the face filiform warts may appear.

Histologically, warts are acanthopapillomas with a marked widening of the interpapillary processes and acanthosis. The horny layer is increased and the cells of the upper layers of the epidermis present a peculiar vacuolization.

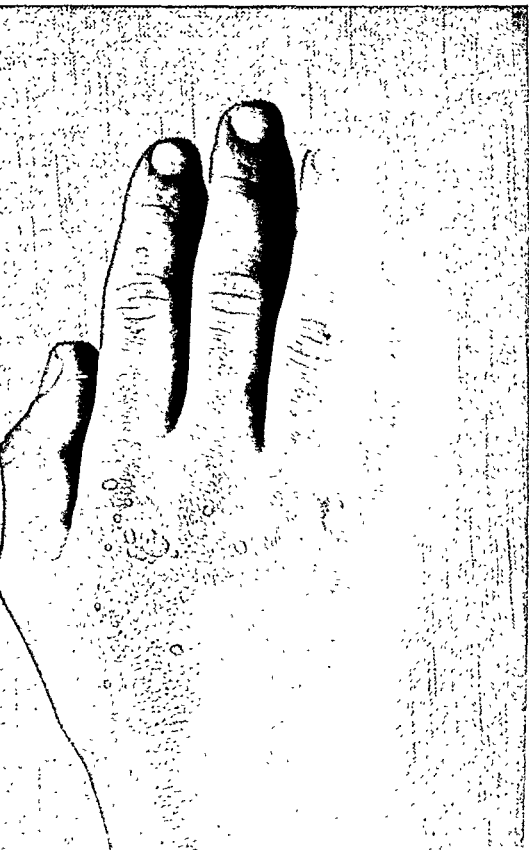
Etiology and pathogenesis Warts are contagious and are probably due to a virus. Autoinoculation is often observed in the form of a linear arrangement of

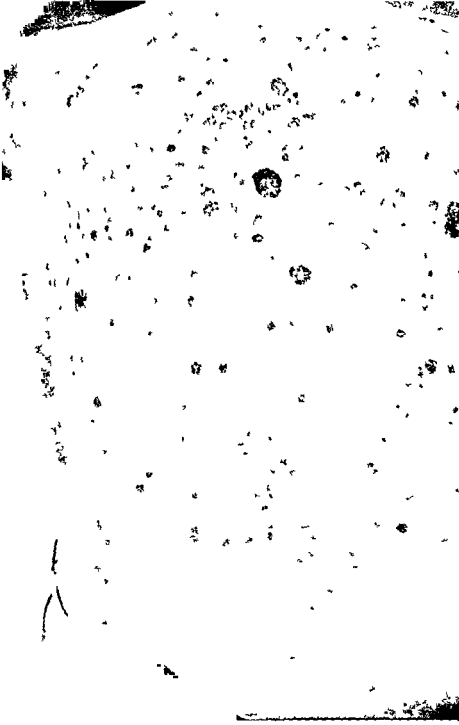


Fig 52 *Herpes zoster* of the middle thoracic region

Fig 53 *Verrucae planae juveniles* (juvenile warts)









56 *Molluscum contagiosum*
There is a slight indentation
center of the larger papules

Condylomata acuminata of the penis



← Fig 56 *Mollis macrospora* There is a slight indentation in the center of the larger papules

Fig 57 *Candelaria acuminata* of the pens



Fig 58 *Dermatitis herpetiformis* There are groups of smaller and larger blisters



small warts along scratch marks or scars Spontaneous cure within a few months or years is the rule.

Treatment Psychotherapy in the form of suggestion therapy is successful in a fair number of cases, especially in juvenile warts (see page 74), the warts are painted with an innocuous colored solution such as eosin or gentian violet. These applications are combined with verbal suggestions that the warts will disappear within a few weeks, or similar techniques may be used. If juvenile warts do not respond to psychotherapy it is preferable to temporize rather than to use surgical methods, which often leave scars. However, surgical therapy is indicated in ordinary warts in the form of curettage and superficial electro- desiccation under local anesthesia. The same treatment may be used in plantar warts of all sizes but x-ray treatment often cures smaller warts on the soles. Liquid nitrogen seems useful for all warts.

Condylomata Acuminata (Acuminate Warts) (Fig 57)

Condylomata acuminata are cauliflower-like, small, papillary tumors and plaques on the penis, vulva and vagina, but are rarely found on the mucous membrane of the mouth. They may be transmitted by sexual intercourse. Sometimes verrucae vulgares and condylomata acuminata caused by the same virus are found in the same patient.

Treatment Cautious application of a 20 per cent podophyllin ointment is the treatment of choice, but this may produce a severe reaction. If this method fails surgical measures under local anesthesia are indicated, i. e., removal of the warts with a diathermy loop or by curettage and electrodesiccation.

Verrucae Seniles (Seborrheic Keratoses) (Fig 55)

Senile warts are flat papules, light brown or black with a greasy verrucous surface, they occur singly or in large numbers on the face, back and dorsa of the hands.

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Molluscum Contagiosum ("Water Wart") (Fig 56)

Molluscum contagiosum (Fig 56) presents pinhead to cherry pit sized, globular, skin colored tumors, often occurring in groups. After a few weeks a simple dissection - the . . .

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histologically there is a wartlike acantho papilloma with liquefaction in the epithelium. Virus particles have been demonstrated in the molluscum inclusion bodies.

Treatment Aureomycin or another antibiotic may be tried topically or internally. Usually surgical measures are necessary, such as (1) incision followed by expression of the molluscum bodies and application of weak tincture of iodine (2) curettage, or (3) superficial electrodesiccation.

Other Virus Diseases

Other virus diseases include the following conditions. Pustulosis varicelliformis (Kaposi's varicelliform eruption) is a superinfection of atopic dermatitis with the virus of herpes simplex occurring most often in infants. Chickenpox (varicella) and smallpox (variola) present skin manifestations but are discussed in medical and pediatric texts. Vaccinia (cowpox) is used for vaccination against smallpox. If such vaccination is carried out in the presence of an existing skin disease such as eczema or psoriasis, a generalized pustular eruption may occur (vaccinia generalisata). This is a dangerous, at times fatal, systemic infection. Therefore smallpox vaccination should not be carried out in the presence of an eczema or any other dermatosis. Milker's nodules and orf (ecthyma contagiosum) are papular and pustular sometimes granulomatous lesions usually found on the hands of farmers who contact infected cows or sheep. Foot and mouth disease may occasionally also affect humans, causing stomatitis and paronychia.

IV. INFLAMMATORY DISEASES OF UNKNOWN ETIOLOGY

Pemphigus Group

Dermatitis Herpetiformis (Dühring's Disease) (Fig 58)

The classic lesions are groups of pinhead sized vesicles on an erythematous base, usually found on the trunk and extremities and sometimes on the face, but rarely on the mucous membranes. There are many variations from this typical picture, erythema, wheals, papules, large vesicles, erosions, excoriations, crusts and residual pigmentation are frequently found. The clinical picture often is pleomorphic. The disease causes burning of the skin and intense itching. Although dermatitis herpetiformis may persist for many years the general health is usually not impaired. Eosinophilia of the blood and of the vesicle fluid is common. The vesicles are subepidermal and there is no acantholysis. Patch tests with potassium iodide may cause a vesicular reaction, apparently not based on an allergic mechanism, and iodides taken internally frequently aggravate dermatitis herpetiformis. The etiology is unknown.

Herpes gestationis is usually considered a form of dermatitis herpetiformis, occurring during pregnancy.

Treatment Sulfapyridine is the drug of choice, having an almost specific action in many cases. Sulfone drugs, such as Fromacetin or Diasone (Dapsone B P C) and Kynex are nearly as effective. Corticosteroids may be used in bullous dermatitis herpetiformis. When secondary infection is prominent antibiotics are valuable. Locally, antiseptic shake lotions and soothing baths give considerable relief (see treatment of eczema pages 133 to 134).

Pemphigus Vulgaris

In this serious and usually fatal disease, medium to large vesicles form on apparently normal skin. Pemphigus often starts with a few bullae and spreads until much of the skin is affected. The mucous membranes tend to be involved, with the production of painful erosions of the mouth. Sometimes the disease begins on the mucous membranes, and skin lesions show up only with later exacerbations. Usually there is no pruritus. The widespread erosions cause much loss of protein resulting in a picture of debility similar to that found in severe burns. Recurrent attacks lead to cachexia and death.

The vesicle of pemphigus is intraepidermal, loss of intercellular bridges or prickles called acantholysis results in the separation of groups of epithelial

cells which can be found on microscopic examination of blister fluid and histologic sections. The loss of intercellular cohesiveness explains why one can detach the upper layer of the epidermis by firm, oblique pressure with one finger (Nikolski's sign).

Pemphigus may take a variety of courses. In pemphigus foliaceus, which resembles an exfoliative dermatitis, scales and crusts replace the blisters. Brazilian pemphigus, an endemic disease, presents the same picture. Pemphigus erythematodes (Senear-Usher syndrome) has morphologic features in common with lupus erythematosus and seborrheic dermatitis. Lesions are located mainly on the face, chest, and back, the course is relatively benign. In pemphigus vegetans hypertrophic granulation tissue tumors form over the erosions.

Treatment. Corticotropin (ACTH) or one of the corticosteroids, alone or in combination, are the treatment of choice. Dosage and dangers of treatment with these drugs are discussed elsewhere (see page 136). In pemphigus very large doses are required, e.g. 100 mg of prednisolone initially, with gradual reduction after a few days to 80, 60, 40 and 20 mg daily, according to the response. When ACTH is used 100 international units (IU) in repository form are given (ACTH Gel or a similar preparation) and the dose is reduced gradually. Sometimes ACTH and corticosteroids are combined. Patients with pemphigus usually tolerate these amounts remarkably well, but one must watch for signs of electrolyte imbalance, infection and demineralization of bone. However, in this otherwise fatal disease it may be necessary to continue corticosteroids even in the presence of undesirable side effects. Potassium chloride is added, 0.3 g four times a day or 1.0 g once a day. Treatment with cortisone and ACTH diminishes resistance to infection, pyoderma, pneumonia and septic conditions have been observed. Therefore, antinfectious therapy with antibiotics or sulfonamides should be given. Although steroids usually produce a more or less prolonged remission in this distressing disease, their ultimate effect has yet to be evaluated. Topically, wet compresses, antiseptic lotions and ointments such as those used in acute eczema (see pages 133 to 134) are helpful. Hospitalization and careful nursing care are essential during the acute phase.

Other Bullous Diseases

There are other bullous diseases which may be discussed with the pemphigus group. The so-called "acute pemphigus" or "butcher's pemphigus" is not true pemphigus but a rapidly fatal, bullous septicemia. Impetigo herpetiformis is characterized by groups of pustules and bullae with severe systemic symptoms. It is associated with hypoparathyroidism and tetany.

Treatment consists of the administration of ACTH, vitamin D and calcium.



Fig 59 *Erythema multiforme* with typical target lesions

Familial Benign Chronic Pemphigus (Hailey and Hailey's Disease)

This consists of serpiginous impetiginoid lesions mainly located on the neck axillae and groins. It appears usually first in young adults, has a benign course but tends to recur. Etiology is unknown. It is not pemphigus but is perhaps related to Darier's disease.

Erythema Multiforme (Erythema Exudativum Multiforme) (Fig 59)

In erythema multiforme the typical lesion consists of a sharply margined erythematous macule or wheal which gradually assumes a violet color although marked edema may cause some lesions to appear white. Vesiculation is common especially in more severe forms. Several rings surrounding each other produce the almost diagnostic target or iris lesions. Papules, blisters, and macular lesions may be present at the same time and produce a polymorphic picture. In acute cases large blisters predominate.

The eruption appears suddenly and is generally symmetric. The extensor surfaces of the extremities, the dorsa of the hands and feet, are most commonly

involved, although the lesions may be generalized. The lips and mouth are often affected and severe cases are accompanied by conjunctivitis, fever and joint pains. Histologically there is a subepithelial perivascular inflammation, the blisters if present are subepidermal. Erythema multiforme occurs in all age groups but most often in young adults.

The following syndromes are either variants of erythema multiforme or probably closely related to it. The Stevens-Johnson syndrome is a sometimes fatal form and pneumonia.

Lesions of erythema multiforme are present on the skin but the clinical picture is dominated by conjunctivitis and erosions of the body openings such as the lips, mouth, nostrils, urethral meatus and anus. In Behcet's disease we find the following triad of symptoms: genital ulcerations, oral aphthae, recurrent attacks of uveitis and iridocyclitis, the skin shows an eruption which resembles erythema multiforme.

Etiology Sunlight is known to provoke erythema multiforme, it often recurs in spring. A virus etiology is suggested by the not infrequent association with herpes simplex and erythema nodosum. Erythema multiforme may follow a cold, focal infection or fatigue, but it is often only a symptom complex which may be initiated by infection, sensitivity to drugs or foods and other factors.

Treatment Mild cases may clear up within a week and require little treatment. In moderately severe cases lotions and ointments are used as in acute eczemas (see pages 133 to 134), internally, antihistamines or salicylates may be tried and niacinamide as a protection against the sun. Mouth washes and compresses are prescribed when the mucous membranes are involved. In severe cases antibiotics or γ -globulin may be tried and even cortisone or ACTH may be needed.

Erythema Nodosum (Fig. 60)

Erythema nodosum consists of round, bright red, tender, slightly raised, subcutaneous nodules which are cherry to plum sized or even larger. Symmetrically distributed usually over the shins, they may also appear on the extensor surface of the arms. Onset is often acute with fever and joint pains and may occur 2 to 3 weeks after an upper respiratory infection. The older lesions gradually acquire a bluish discoloration. In general the lesions are discrete and vary in number from 4 to 30 but if numerous they may fuse to form large, indurated areas. Several successive crops of lesions are the rule, the disease lasting several weeks or many months. It is seen most frequently in young women. Histologically there is a perivascular inflammation according to Miescher, groups of epithelioid cells arranged in radial fashion and surrounded by lymphocytes are characteristic for the disease.

Fig. 60 *Erythema nodosum* There are bright red coalescent nodules on the lower legs with a more diffuse infiltrate over the right shin



- Fig 61 *Chronic discoid lupus erythematosus* Red circumscribed lesions with adherent partly follicular scales Large lesions show central atrophy and depigmentation

Fig 62. *Chronic discoid lupus erythematosus* Note the follicular pattern of the hyperkeratosis

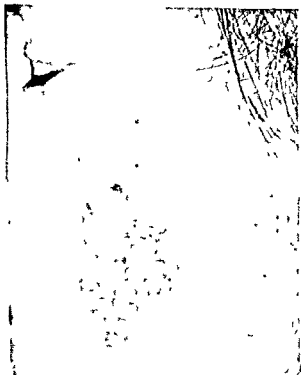
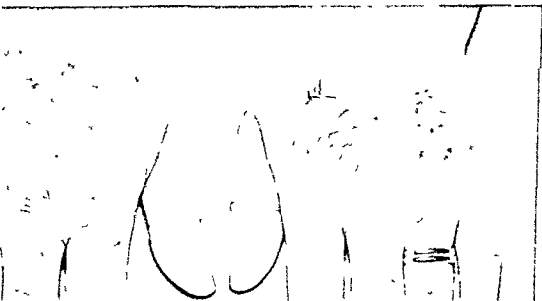


Fig 63 *Acute lupus erythematosus* Sharply margined erythema with barely detectable scaling



Fig 64 *Acute lupus erythematosus* Red, slightly elevated and hyperkeratotic lesions of dorsa of hands



tiology of erythema nodosum is unknown. It is probably a disease in which occurs during or after certain infections, in children especially it is associated with tuberculosis and rheumatic fever. In adults the onset more often follows a streptococcal sore throat or tonsillitis. Erythema nodosum has also been observed in trichophytosis, lymphogranuloma inguinale, coccidioidomycosis and gonorrhea.

Wet compresses, elevation of legs, and use of lotions diminish the local discomfort. Antibiotics and sulfonamides are without effect, but salicylates often help. Cortisone or its newer derivatives are very effective in severe cases.

Lupus Erythematosus

This disease occurs in two forms. One is the rather common, benign, chronic discoid lupus erythematosus, confined essentially to symptoms of the skin, the other, a relatively rare and serious form is acute lupus erythematosus, with general and systemic involvement.

Discoid Lupus Erythematosus (Figs. 61 and 62)

The early manifestations of discoid lupus erythematosus are red, coin sized, slightly infiltrated lesions which are usually found on exposed skin, especially on the nose, the "V" of the neck and the back of the hands. At times the lesions on the cheeks and nose coalesce to assume a "butterfly" shape. Scaling of the follicles is common.

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erythema. As a result the ears and alae nasi may be involved and undergo mutilating changes resembling those of lupus vulgaris.

The disease is chronic and recurrent. It is often aggravated by sunlight and it is more frequent in countries with a sunny climate.

Acute (Systemic, Disseminated) Lupus Erythematosus (Figs. 63 and 64)

Acute lupus erythematosus usually starts as such but may occasionally begin as the discoid form. In acute lupus the face and the dorsa of the hands are



Fig 66 *Lichen planus*
Flat topped, polygonal papules becoming confluent in one area, and tiny follicular papules in the periphery

Fig 67 *Lichen planus* The violaceous color is very typical. A whitish hyperkeratosis is visible on the surface of the papules. Most individual papules have fused to form larger lichenified patches.





Fig. 68 *Lichen planus* of the posterior portion of the buccal mucosa



Fig 69 *Psoriasis vulgaris*



Fig 70 *Psoriasis vulgaris* After removal of the parakeratotic scale a red slightly indurated lesion appears with bleeding points (Auspitz's sign)

Fig 72 *Psoriasis vulgaris* Sharply demarcated lesions with whitish scales on the hairline and ear



Fig 73 *Guttate parapsoriasis* with slightly scaling red papules



Lichen Planus (Figs 65 to 68)

The primary lesion is a highly characteristic pinhead sized, flat, polygonal, sharply demarcated papule. The surface of large plaques often has a waxlike sheen and on close examination grayish puncta or streaks (Wickham's striae) may be seen. At first the lesions are bright red later changing to a rather typical violet color and finally to brownish red. Dark pigmentation often persists for a long time after the lesions have cleared. Although the flexor surfaces of the arms, the lower legs and the genitals are especially favored by localized forms of this disease, any part can be involved and the disease may be generalized. Confluence of lesions forming patches of varied sizes and shapes is common and the skin may become infiltrated and lichenified. The lesions are covered with an adherent whitish or brownish hyperkeratosis. ✓

There are some special forms of lichen planus. Lichen planus annularis, in which ring shaped forms are prominent, is seen especially on the penis and scrotum. Lichen planus verrucosus describes a variant with hypertrophic lesions which is most frequent over the shins. Lichen planus et acuminatus trophicans presents, besides typical lesions round plaques with central atrophy, on the

where is mucous membrane involvement of the mouth, tongue, lips or vagina. Here one sees a whitish, papular hyperkeratosis which usually coalesces to form a lacy network, or plaque-like lesions. /

Lichen planus usually persists for several months, often longer. Crops of new lesions after intervals of several months or years may occur, especially in older people. Acute exanthematic forms and circumscribed lesions on the arms have a tendency for spontaneous recovery. Verrucous lesions of the lower legs and mucous membrane patches are notoriously chronic and persistent.

The histologic picture is characteristic. The epidermis shows hyperkeratosis and acanthosis, the interpapillary processes are enlarged, in the dermis there is a dense infiltrate of lymphocytes which is sharply limited to the upper part of the cutis.

The etiology is unknown but an infectious agent is suspected. Provocation of lichen planus by emotional stress and nervous tension, also by arsenic medication, is known. External trauma may influence the localization, this is seen in more intense involvement of areas exposed to sunlight and in linear lesions following scratching.

Treatment In generalized cases rest, reassurance, relief from worry, and the use of mild antipruritic lotions or ointments (see pages 133 to 134) are usually all that is needed. Generous amounts of vitamin B-complex with vitamin C are

helpful In localized forms superficial x ray treatment is the treatment of choice In refractory cases bismuth salicylate may be given the dose is 1 cc intra muscularly twice a week for a total of 12 to 15 injections Arsenic is a time honored drug usually prescribed as follows Fowler's solution 10.0 and aqua menthae piperitae q s ad 30.0 To begin with 5 drops three times a day after meals are taken with water milk or tea The dose is increased by 1 drop three times a day until 20 drops three times a day are reached The prescription should not be refilled more than three times because of the danger of arsenic keratoses and carcinomas In case of intolerance to oral arsenic (anorexia heartburn diarrhea) injectable organic or inorganic arsenic preparations may be used

The hyperkeratotic verrucous lesions of the lower legs often require energetic topical treatment including keratolytic ointments tar ointments 30 per cent trichloroacetic acid and electrodesiccation

Two unusual possibly related diseases may be mentioned at this point In lichen nitidus numerous whitish or slightly pigmented papules are seen usually on the penis although they may be found elsewhere Lichen albus or lichen sclerosus et atrophicus is characterized by white papules Sclerosis of the dermis an infiltrate of lymphocytes and early destruction of elastic fibers are seen

Psoriasis (Figs 69 to 72)

The primary lesion of psoriasis is a coin sized sharply demarcated papule covered with heavy whitish scales Removal of the massive deposit of parakeratotic scales reveals an infiltrated macule with bleeding points on its surface The bleeding points represent traumatized capillaries in the greatly elongated dermal papillae The main part of the psoriatic lesion consists of heaped up scales and when these are less extensive the surface appears red Gentle scraping produces a whitish surface since the hyperkeratosis is easily loosened and rendered opaque

Psoriatic plaques may be found anywhere on the body but the knees elbows and scalp are favored sites (Figs 70 and 71) No loss of hair occurs with scalp lesions Guttate psoriasis with numerous small lesions is a form which often follows an upper respiratory infection At times psoriasis may affect the flexural instead of the extensor surfaces and the anus groins and axillae (psoriasis inversa) In pustular psoriasis a rare variant pustules appear usually on the palms and soles or there are acute febrile attacks with disseminated pustules Psoriasis may progress into a generalized erythroderma (psoriatic erythroderma) When the nails are involved they become stippled discolored and deformed In arthropathic psoriasis there is also rheumatoid arthritis like deformity of the joints especially the terminal phalangeal articulations

Course—Psoriasis may begin at any age and usually remains an undesired

companion for life, with recurrent exacerbations and remissions. Clearing of lesions is usually incomplete but complete disappearance of the disease for many months and even years may occur. Psoriasis is a benign disease except for the rare pustular, arthropathic or erythrodermic forms.

Etiology The cause is unknown. Emotional and nervous stress may cause aggravation. The tendency of this disease to run in families suggests some sort of metabolic defect, but intensive investigation has failed so far to elucidate causative metabolic factors or infectious agents.

Treatment The patient should be reassured about the harmlessness of the disease but also forewarned to expect recurrences. Topical treatment is pre eminent. Salicylic acid, ammoniated mercury and tar are the main drugs for ambulatory patients. Scales are removed with ointment containing 5 per cent salicylic acid in yellow petrolatum. Afterwards one of the following ointments is used twice daily.

Ammoniated mercury, 5 per cent, and anthrasol, 5 per cent, in equal parts of cold cream and white Vaseline or 5 to 10 per cent liquor carbonis detergens in either a washable base or yellow petrolatum. Sorsis Alpha and Sorsis Beta (Ar-Ex Cosmetics) and Pixeyl and Psorox (Genatosan) are good proprietary anti-psoriatic preparations.

When more intensive treatment is needed, crude coal tar, chrysarobin or Anthralin are used. Because of their staining properties only exceptional patients will use them at home. The Goeckerman treatment consists of application of a 5 per cent coal tar ointment at night and an ultraviolet treatment the next morning utilizing the photosensitizing capacity of tar. Chrysarobin is used in yellow petrolatum 0.2 to 3 per cent, or in chloroform, 0.5 to 5 per cent. Because it stains its use is usually restricted to hospitalized patients. Treatment is continued until a moderate degree of skin irritation has been reached, then a bland ointment is applied until the reaction has subsided. Anthralin (Dithranol B.P.) ointments 0.1 to 1 per cent, have a similar effect but are less messy. As both drugs may cause conjunctivitis they are not recommended for psoriasis of scalp and face. In psoriasis of the scalp 5 per cent salicylic acid in a washable cream or in Carbowax 400 helps to remove the scales, 10 per cent liquor carbonis detergens in Topisol (Texas Pharmacal Company) is used afterwards. Siroil, a proprietary antipsoriatic drug, is often helpful in scalp lesions. These preparations are welcomed by the patient because they can easily be removed by shampooing. Internal treatment of psoriasis is usually disappointing. Soybean lecithin in the form of Granulestin is recommended. Corticosteroids are indicated mainly in arthropathic psoriasis and in psoriatic erythroderma. The newer derivative triamcinolone (Anistocort, Kenacort) is very effective in psoriasis. However, the disease usually recurs after the drug has been discontinued.

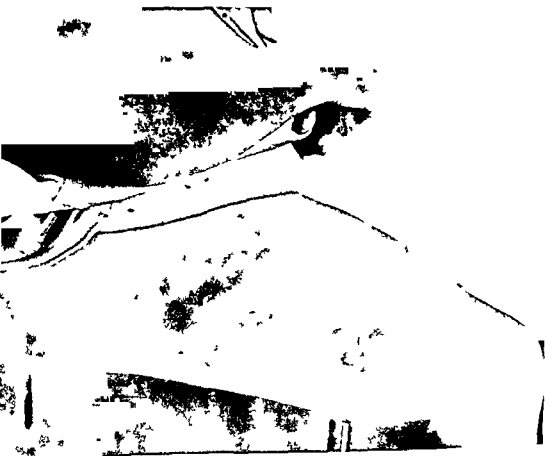
Diseases Resembling Psoriasis

There are several rare skin diseases which at times resemble psoriasis but probably are not related to it

Guttate parapsoriasis (pityriasis lichenoides chronica) (Fig 73) consists of an eruption of small reddish and brownish papules on the trunk and extremities. Individual lesions resemble psoriasis, lichen planus and pityriasis rosea the eruption as a whole may look like secondary syphilis. The disease is eminently chronic but usually without subjective symptoms. It is very resistant to treatment but ultraviolet light and vitamin D may help.

Parapsoriasis en Plaque (Brocq's Disease Xanthoerythrodermia perstans) presents circumscribed patches of red or yellowish red color and of varying size with superficial scaling (see Fig 74). Subjective symptoms are usually absent. There is good evidence that many cases of this disease represent early

Fig 74 Parapsoriasis en plaque xanthoerythrodermia perstans. Brownish red patches with slight infiltration.



stages of mycosis fungoides or other reticuloendotheliomas. No effective treatment is known.

In acrodermatitis continua of Hallopeau pustules occur on the fingers and are followed by atrophy and loss of nails. At times this condition cannot be distinguished from pustular psoriasis.

In pityriasis rubra pilaris there are follicular, pointed hyperkeratotic papules situated on the extensor surfaces of arms and legs, especially of fingers and toes. The course of the disease is chronic.

Treatment. Mild antipsoriatic ointments may be used. Vitamin A in large amounts is helpful in some cases.

Pityriasis Rosea (Fig. 75)

In this self-limited skin disorder, oval patches 0.5 to 3 cm in diameter develop on the trunk, neck and proximal parts of the extremities. The long axes of the plaques tend to run parallel with the lines of cleavage of the skin. Pinkish-yellow in color, the lesions have fine scales which are attached centrifugally (collarette). In about one-half the cases a single primary plaque, the herald patch, appears 1 to 3 weeks before the onset of the generalized eruption. Itching is usually absent but at times is troublesome. The lesions disappear within 3 to 10 weeks, recurrences are very rare. The diagnosis is aided by the typical location and by the course of the disease. Pityriasis rosea usually can be distinguished from seborrheic dermatitis, the latter is found in addition, in the folds of the body and on the scalp, and has a chronic recurrent course. The etiology is unknown. An infectious agent is suspected.

Treatment. Treatment is indicated only when warranted by subjective symptoms. Mild antipruritic lotions containing 2 per cent salicylic acid or 1 per cent phenol may be tried. Hydrocortisone ointments relieve discomfort and seem to speed up healing. Internal antihistamines help when the pruritus is more pronounced. Ultraviolet irradiation often shortens the course.

Miscellaneous Granulomas

Granuloma Annulare

Granuloma annulare is made up of multiple, deep-seated, whitish papules which are usually arranged in a circular manner. Although commonly found on the dorsa of hands, feet, elbows or neck, it may be located anywhere.

The disease

it progresses

character

etiology is unknown

—degeneration of collagen surrounded by lymphocytes. The

Treatment Lesions often disappear following a biopsy or irradiation with ultraviolet rays or x-rays, but they may recur

Necrobiosis Lipoidica

The early lesions are small, reddish plaques with yellowish centers. Gradually central atrophy occurs and gives a typical waxy look to the middle of the lesion. The shins are sites of predilection. The etiology is unknown. The disease occurs usually in young women and mostly, but not invariably, in diabetics. Although the lesions are chronic and usually asymptomatic, occasionally they may ulcerate.

Granulomatosis Disciformis (Miescher)

This condition consists of sharply circumscribed, slightly infiltrated, reddish lesions probably representing a "nondiabetic necrobiosis lipoidica".

Reticulosis

This term covers those rare granulomas with and without lipoid infiltration such as Letterer-Siwe disease, Hand-Schüller-Christian disease, and eosinophilic granuloma of bone (with skin lesions). They are closely related diseases of the reticuloendothelial system occurring in children. Skin eruptions, bone defects and hepatosplenomegaly are observed. These diseases are now called *histiocytosis X*.

Lymphocytoma

Bluish or brownish small subcutaneous tumors are found on the lobes of the ears, the face, the genitals and occasionally in other locations. Histologically they consist of dense lymphocytic infiltrates with germinal centers. The lesions may heal spontaneously. The etiology is unknown. According to Pachoud it is a transmissible disease.

Treatment Lymphocytoma may respond to x-ray, radium treatment, or penicillin injections. Excision at times is the simplest measure.

Sclerodermas and Atrophies

Localized Scleroderma (Morphea) (Fig. 76)

The characteristic lesion is a circumscribed patch with atrophy of the epidermis and sclerosis of the cutis, usually surrounded by a violaceous border. Blood vessels can be seen shimmering through the atrophic, hairless areas. Any area of skin may be involved. In the scalp alopecia results. The shape of the lesions varies a great deal, they may be single or several in number from coin

sized to large, palm sized plaques, a special form is scleroderma "*en coup de sabre*", with bandlike lesions and scarlike atrophy of the connective tissue. Without treatment the lesions usually grow and may after several years cover a large part of the skin. Since the atrophic epidermis is easily traumatized, blisters, infection and ulceration may occur. Spontaneous regression has been observed.

Treatment Some observers have reported good results following treatment with antibiotics. 10 to 20 injections of 600 000 units of penicillin are recommended or broad spectrum antibiotics such as Aureomycin, Chloromycetin, or Terramycin may be given for 8 days (250 mg four times daily). Progression is halted, the inflammatory violaceous ring disappears quickly, and the sclerosis regresses slowly over a period of months and years although the atrophy persists. X ray or thorium X treatment also seem helpful.

Generalized (Diffuse) Scleroderma (Sclerodactylia) (Fig. 77)

The disorder may begin insidiously like acrocyanosis or Raynaud's disease. More or less gradually the skin of the fingers becomes sclerotic and bound down to the underlying structures so that the fingers can hardly be moved. The epidermis is tightly stretched and thin. Ulcerations of the fingertips develop and the terminal phalanges may disappear gradually to give the hands a clawlike appearance. There is a tendency to chronic paronychia (Fig. 77). The sclerotic process extends from the hands to the wrists and arms and the joints become stiff. As the skin of the cheeks, lips and lids becomes drawn the face assumes a masklike appearance. Stellate telangiectases, depigmentation and hyperpigmentation may occur. Painful nodules with calcification may appear around the involved joints and may ulcerate (syndrome of Thibierge-Weissenbach). In occasional severe cases the sclerosis involves large parts of the trunk. Diffuse scleroderma often also involves the mucous membranes of mouth, pharynx, larynx, esophagus and stomach. The disease causes a characteristic pulmonary fibrosis with respiratory difficulties and also pathologic changes of the heart and brain. Inflammatory processes leading to atrophy occur in the muscles of the arms and less frequently, the legs, such cases may represent a transition to dermatomyositis.

Generalized scleroderma often is a progressive fatal disease, discomfort and restriction of mobility lead to incapacity. At times the condition remains stationary for many years with relatively minor changes of the skin of the face and hands. The histologic picture of the various forms of scleroderma is rather uniform. The epidermis is thinned, the papillae are flattened out and the adnexa of the skin have disappeared. The collagenous connective tissue is swollen and homogenized, there is perivascular inflammation.

The etiology is unknown. In analogy to lupus erythematosus a systemic

infectious allergic disease of the connective tissue is assumed originating from the blood vessels, hence the common term "collagen" diseases

Treatment No effective therapy is known. Antibiotics may be tried, but they have little effect. Measures and drugs improving circulation and producing vasodilation are of limited help. Sympathectomy produces at best temporary improvement. Cortisone and ACTH are usually only palliative. Chelating agents have been recommended. The patients should be advised to move to a warm climate.

Rare Related Diseases

The following rare diseases may be discussed with scleroderma. Scleredema of Buschke shows progressive induration and a doughy swelling of the skin and subcutaneous tissues. The affection may start on the neck and extend to the face and trunk. Its onset usually follows some febrile disease. Poikiloderma of Jacobi presents a mixture of inflammatory and scleroderma like changes of the skin. There is a pleomorphic picture with redness, scaliness, atrophy, sclerosis and pigmentary changes, at times resembling chronic radiodermatitis. Dermatomyositis is a severe systemic, often fatal disease. The skin lesions are polymorphic, sometimes resembling erythroderma, sometimes systemic lupus erythematosus. Atrophy often follows. There is extensive inflammation of the muscles, with subsequent atrophy and contractures.

Treatment Corticosteroids and ACTH may produce marked remissions.

Acrodermatitis Chronica Atrophicans (Herxheimer)

The primary lesions are palm sized, slightly infiltrated, bluish red plaques on the extremities, with some edema. Sometimes the lesions are in the form of bands which extend downwards from the elbows. On the feet and legs the lesions increase in size and may spread over large areas of the thighs or lower legs. Gradually atrophy appears and the skin takes on a thin and wrinkled appearance that has been likened to crumpled cigarette paper, the blood vessels become visible through the thinned epidermis. Fibrous, painless subcutaneous nodules may appear in the region of the elbows. The etiology is unknown; an infectious agent is suspected.

Treatment The acute symptoms are improved by antibiotics, their effect being similar to that obtained in circumscribed scleroderma (see page 107).

Other Rare Idiopathic Atrophies

Atrophoderma vermiculata is a symmetric eruption of the cheeks consisting of small, individual pitlike atrophic areas. It occurs mostly in young persons, occasionally it follows acne vulgaris. Anetoderma of Jadassohn consists of small, spotty atrophy without apparent preceding inflammation.

V. ALLERGIC SKIN DISEASES

Allergic skin diseases are due to a sensitivity to usually harmless substances. Contact with these substances may occur externally, or they may be introduced by mouth, by inhalation, or by injection. The clinical picture depends on the layer of the skin in which the allergic reaction occurs. When the epidermis is the shock organ an eczematous dermatitis occurs. When the vascular system of the dermis is involved an urticaria may develop. When the blood itself is the shock organ the allergic process may cause purpura through a decrease in platelets, or agglutination of leukocytes which results in agranulocytosis. Proper test methods are necessary to find the causative agent. In contact dermatitis the test substance is applied as a patch test, in urticaria the allergen is brought in contact with the blood vessels of the corium by scratch or intradermal tests. There are no practicable tests available for purpura and agranulocytosis. Ingestion of the causative agent in patients with purpura produces a fall in thrombocytes in agranulocytosis leukopenia. In infantile eczema and in atopic dermatitis scratch and intradermal tests indicate hypersensitivity of the dermal blood vessels although especially in atopic infantile eczema epidermal changes dominate the clinical picture. The shock organs in allergic diseases and the test methods are presented in Table 2.

Our concepts about allergic phenomena are based to a great extent on animal experimentation. Injection of serum from other animals produces a high degree of hypersensitivity in rabbits and guinea pigs. Reinjection of the serum after a suitable incubation period produces a severe or fatal shock in cases of intravenous injections or a localized hemorrhagic inflammation (Arthus phenomenon) following intradermal injections. This form of hypersensitivity is called anaphylaxis. The serum of the hypersensitive animals contains specific antibodies. In man however specific antibodies can be demonstrated only in some allergic diseases. In urticaria bronchial asthma allergic rhinitis, atopic dermatitis passive transfer of the sensitivity can be achieved by means of the Prausnitz-Kustner test. The serum of the donor is injected intradermally into the recipient. After 24 hours during which time the antibody becomes fixed to the skin injection of the allergen into the prepared site produces a positive reaction. Accidental transfer of the cutaneous vascular form of sensitivity has been observed in cases of blood transfusion in In contact dermatitis the an although there is very good evidence that they are transmitted by lymphocytes. However the phenomena of sensitization the incubation period in experimental sensitization to contact allergens and the specificity of hypersensitivity can be considered proof of the allergic nature of contact dermatitis.

Table 2
Shock Organs and Test Methods in Allergic Diseases

<i>Disease</i>	<i>Shock Organs</i>	<i>Tests</i>	<i>Demonstration of Circulating Antibodies</i>	<i>Desensitization</i>
Allergic contact dermatitis	Epidermis (also dermis)	Delayed reaction to patch tests (and intradermal tests)	Not possible	Usually not feasible except in plant dermatitis
Urticaria	Vascular system of dermis	Immediate reaction to scratch or intradermal tests	Possible	Not very practical
Atopic dermatitis, infantile eczema	Vascular system of dermis	Immediate reaction to scratch or intradermal tests	Possible	Helpful only at times
Thrombocytopenic purpura	Blood and Capillaries	Thrombocytopenia following ingestions of allergen	Possible	Not feasible
Drug eruptions	Cutis	Delayed or immediate reactions to intradermal tests, thrombocytopenia	Rarely possible	Usually not feasible

It is generally believed that the union of antigen and antibody releases histamine or histamine-like substances which elicit the allergic reaction. The older term, "idiosyncrasy", actually means the same as allergic hypersensitivity and is used at times to indicate unexpected, severe degrees of hypersensitivity.

For qualification as an allergic disease, the following criteria should be fulfilled, although in reality not all can be demonstrated in every instance.

1. High degree of hypersensitivity
2. The reaction must be qualitatively different from the normal pharmacologic action of the causative agent
3. The clinical picture has to correspond to the shock organ and not to the chemical properties of the chemical agent.

- 4 Specificity of hypersensitivity
- 5 Demonstration of development of sensitization
- 6 Demonstration of antibodies

Here is an example for principles 2 and 3. When the shock organ is the epidermis a contact dermatitis always ensues, regardless of whether the agent is a drug turpentine or cement. When the vessels of the cutis form the shock organ urticaria results, no matter whether the antigen is a food, a drug, or ultraviolet light. Desensitization usually is possible only in diseases based on cutaneous vascular hypersensitivity, and in contact dermatitis from plants.

Eczema—Dermatitis

Acute Eczema or Dermatitis

This is mainly a disease of the epidermis, the dermis being involved mainly secondarily. Histologically, eczematous dermatitis shows a spotty intercellular edema in the epidermis called spongiosis. This is accompanied by dilation of blood vessels, a lymphocytic infiltrate of the adjoining parts of the dermis, and migration of lymphocytes into the epidermis (Fig. 78). The clinical picture varies with the degree of inflammation. If there is only moderate spongiosis present macroscopically pinhead sized disseminated papules appear more or

Fig. 78 Microscopic picture of eczema. The epidermis shows intraepithelial spongiosis and vesicles and the cutis a lymphocytic infiltrate. Lymphocytes have migrated into the epithelium and the spongiotic cavities.



less red according to the degree of vascular dilation. Greater intraepithelial edema leads to small vesicles and eventually to large blisters. When the vesicles and blisters rupture the condition is a weeping eczema (Fig. 79). The epidermis tries to repair the defects, the prickle layer becomes thickened and the process of keratinization is so much accelerated that a parakeratotic layer is formed which still contains nuclei. The clinical manifestations of these changes are scales, crusts and thickening of the skin. ✓

Chronic Eczema or Dermatitis

Eczematous dermatitis often recurs in the same location, the chronic inflammation resulting in lichenification. An eczema may also begin as a lichenified dermatitis which indicates the skin is thickened, the minute skin wrinkles are accentuated and the skin is divided by deep lines into a chessboard-like arrangement. The lesions may show different stages of development at one time; they may be disseminated or they may become confluent. In extreme cases the whole skin may be involved as an erythroderma. Besides papules and lichenified plaques, vesicles, weeping, scaling and crusting lesions may occur.

✓ Eczemas are usually very pruritic. Resulting complications are excoriations, secondary infection with folliculitis and even furuncles, lymphadenitis and lymphangitis.

Classification of Eczemas

While it is possible to describe the eczemas from a purely morphologic view point, a classification of eczemas according to etiology and pathogenesis is more helpful, although much of this is still hypothetical.

Three main groups of eczemas are distinguished: (1) contact dermatitis, (2) seborrheic and microbial (parasitic) eczemas, and (3) atopic dermatitis (neurodermatitis).

Contact Dermatitis (Figs. 80 to 89)

Contact dermatitis is due to contact of the skin with external substances. They usually contact the skin directly from the outside but may also reach it indirectly from the inside following ingestion, inhalation or injection. Contact dermatitis is usually an allergic phenomenon but primary toxic irritation plays an additional role, both in the sensitization and in provocation of the eczema. Damage by alkaline materials such as soap, soda or cement facilitates sensitization to turpentine, potassium bichromate and nickel (Burchhardt). Secondary invasion of the dermatitis with staphylococci and other bacteria leads to formation of pustules, microabscesses and local recurrences (Miescher, Storck). In occupational dermatitis the hands and forearms are affected or the face when volatile and powdery substances are the offenders. Contact dermatitis from

Fig. 79 *Bullous weeping dermatitis* after the application of Pell-dol (scarlet red) ointment

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Fig 79 *Bullous weeping dermatosis* after the application of Pell doli (scarlet red) ointment



Fig 80 *Acute contact dermatitis* from the nickel parts of garter straps and from a bracelet containing nickel. Note spread of eczematous lesions to other parts of the body

Fig 81 *Photoallergic (photocontact) dermatitis* after application of an ointment containing sulfanilamide



Fig 82 *Positive patch test*, with nickel coin showing erythema and eczematous papules



Fig 83 *Acute contact dermatitis* of the face from hypersensitivity to a mouth wash with diffuse and patchy redness and infiltration



Fig 84 *Positive patch test with swelling and vesiculation in patient allergic to a mouthwash*



Fig 85 *Contact dermatitis following application of a powder that contained resorcin*

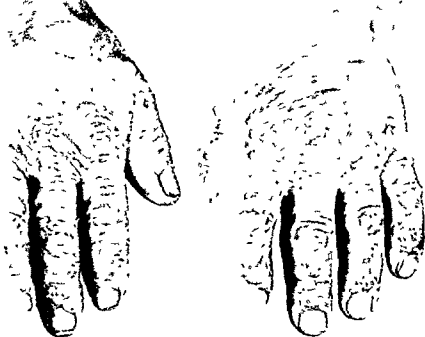


Fig 86 Occupational eczema of a mason. There is lichenification and scaling of the dorsa of hands and fingers with erosions and fissures

Fig 87a Occupational eczema of a female worker due to sensitivity to potassium dichromate used in dyes and mordants

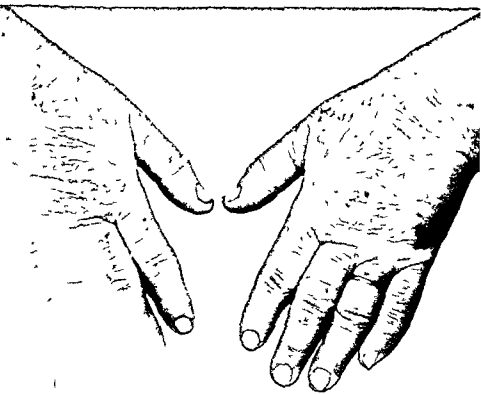




Fig 87b *Strongly positive patch test with potassium dichromate in worker handling dyes*

clothing shoes, girdles, garters, brassieres and bracelets (Fig 80), occurs at

bacteria

The cause of contact dermatitis may be found by patch tests. The suspected substances are applied to the skin in proper concentrations (for technique, see pages 255 and 259) and are left there for 24 hours. A delayed reaction in the form of a localized dermatitis constitutes a positive patch test.

The following is a list of common contact eczemas.

Contact dermatitis from medication Eczematous sensitization of the skin usually occurs after repeated contact with the medication. Drugs such as anesthetics, antiseptics and mercurial preparations are very frequent culprits. Contact dermatitis is often found in patients suffering from chronic skin diseases such as pruritus ani, stasis dermatitis, infectious eczema and psoriasis. It is advisable in these instances to test a few medications with a patch test before using it. Antibiotics such as penicillin, streptomycin or neomycin, and antiseptic drugs may produce contact dermatitis, in sensitivity to procaine penicillin, the procaine frequently is responsible. Sulfonamides at times cause contact dermatitis, and occasionally photocontact dermatitis (photoallergy) (Fig 81), which is due to sensitivity to the combined effect of light and sulfonamide (Epstein, Burckhardt). Every drug apparently can cause contact dermatitis, but some are more likely to do so than others.

Contact dermatitis from clothing Finishes or dyes in clothing, underwear, socks or stockings, furs, or the elastic of garters, girdles or brassieres may

especially in women (Fig 80), the specific sensitivity can be demonstrated by a patch test with nickel sulfate or a nickel coin (Fig 82).

Contact dermatitis from cosmetics Face cream, powders, hair oil and shampoos are common offenders. The eyelids are frequently involved because of their tender skin. *p*-Phenylenediamine, the base of many hair dyes, is a potent sensitizer. Nail polish containing synthetic resins affect especially the face and eyelids. Mouthwashes may be the cause of perioral dermatitis (Figs 83 and 84).

Occupational dermatitis (Figs 86 to 87) In occupational eczemas toxic irritation plays an important part because of organic solvents such as gasoline and benzene or alkaline agents such as soaps, soda or lyes. Sensitivity to contactants develops simultaneously or later. Besides the classic signs of eczema there are also scaliness, fissures, paronychia and pustules.

The following are common examples of occupational dermatitis. Eczema of masons is caused by contact with cement and mortar which are strongly alkaline and first produce a toxic irritation. Persons with decreased alkali resistance are especially prone to irritation of the skin (For test for alkali resistance, see page 257.) Afterwards an allergy to potassium bichromate develops traces of which are found in cement (Jaeger and Pelloni). In house wife's eczema the skin is damaged by contact with soaps and synthetic detergents, here, too, the reduced alkali resistance may play a role. Later on sensitivity develops to turpentine, floor wax, nickel or chromate. Eczemas from nickel sulfate and chromate are observed in persons engaged in nickel and chrome plating industries. Dyes, synthetic resins, formalin and explosives are further examples of substances which may cause occupational dermatitis. A complete listing of occupational contacts will be found in the textbook *Occupational Diseases of the Skin* by Schwartz, Tulipan and Birmingham (see Bibliography).

Contact dermatitis from plants. Contact dermatitis from the Japanese prim rose, especially *Primula obconica*, is a classic example of plant dermatitis. Bloch succeeded in sensitizing experimentally all test persons with a con-



Fig 88 *Poison ivy contact dermatitis showing linear and bullous lesions* (Marshfield Clinic)



Fig 89 *Occupational eczema* in a barber with hypersensitivity to nickel. The eczema was provoked by scissors that contained nickel.

Fig 90 *Seborrheic dermatitis* over sternum

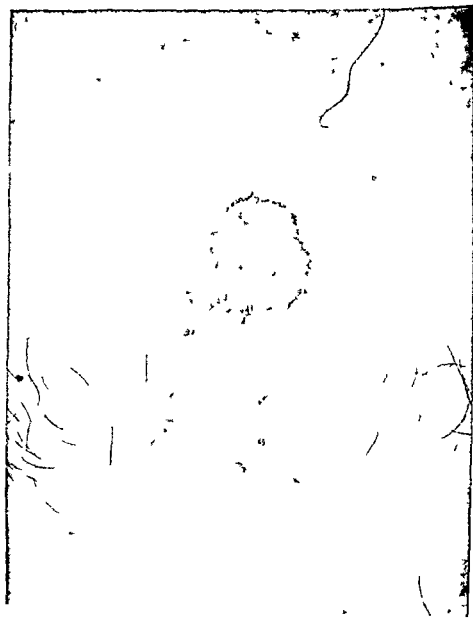




Fig 91 *Seborrheic dermatitis* with diffuse redness and scaling of the ears and cheeks

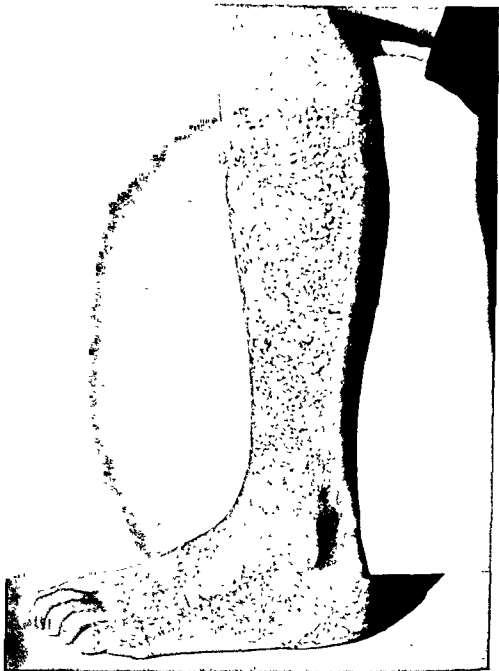


Fig 92 *Eczema of lower leg (stasis dermatitis)*, probably of bacterial origin, with diffuse redness, scaling and infiltration

centrated extract of this primrose and thus proved that everybody can be sensitized. In the United States the most potent and frequent sensitizers among plants are the members of the *Rhus* species such as poison ivy, poison oak and poison sumac (Fig. 88). Weeds, especially short ragweed, are a frequent source of contact dermatitis. Contact dermatitis from weeds is due to the so-called oleoresins, the causative agents being the ether and alcohol soluble fraction of the plant and not its proteins. The rinds of oranges and lemons may cause occupational contact dermatitis. Turpentine, the resin of a pine species, is a common solvent and one of the most frequent causes of contact dermatitis in housewives, as it is contained in floor wax and shoe polish. It also causes contact dermatitis among painters and others who use turpentine to clean their hands. The oxidation products of turpentine are the real sensitizers (Hellestrom).

Seborrheic and Microbic (Parasitic) Dermatitis (Figs. 90 to 92)

These eczemas are characterized by special clinical pictures. The microbial origin is generally assumed and often, but not always, proved.

Classic seborrheic dermatitis is different from contact dermatitis. The lesions of typical seborrheic dermatitis are coin shaped, sharply demarcated, yellow brown in color, and covered by fine scales. Vesicles do not occur. Typical locations are the skin over the sternum, between the shoulder blades, the axillae and the groin. Less sharply demarcated, somewhat indurated plaques are found behind the ears, in the external ear, on the scalp, and on the face. In the intertriginous areas, the axillae, the groin, and behind the ears, a weeping dermatitis often develops. On the scalp and in the eyebrows, seborrheic dermatitis can lead to a slowly progressing alopecia. In the nuchal region, lichenified patches may develop. Occasionally seborrheic dermatitis progresses to become a generalized erythroderma. Seborrheic dermatitis is a constitutional disorder which comes and goes, often over many years. The French classify this affliction as *eczémaides psoriasiformes*.

Mild forms of seborrheic dermatitis are diffuse pityriasis simplex of the scalp and the circumscribed pityriasis alba of the face. This condition produces fine scaling without redness.

There are gradual transitions between seborrheic dermatitis and the so called nummular eczema. Nummular eczema consists of sharply circumscribed, red, round patches of papules and vesicles which may be weeping or lichenified. They resemble ringworm and therefore they also have been called mycotic or parasitic eczema. The main locations are the hands and feet, less frequently the trunk. The etiology of nummular eczema is not clear but possibly staphylococci.

or other microbes of the skin play a role and often there is an atopic background. A fungus infection may be excluded by microscopic examination and by a negative trichophyton reaction.

Eczema of the external ear, of the anogenital region, and of the lower legs also may be grouped with the microbial (parasitic) eczemas, but other factors besides bacterial invasion usually play a role.

Atopic Dermatitis (Disseminated Neurodermatitis) (Figs 93 to 96)

Atopic dermatitis has many synonyms: disseminated neurodermatitis, Besnier's prurigo, disseminated lichen Vidal, late exudative eczematoid, endogenous eczema, constitutional eczema, flexural eczema.

Atopic dermatitis has a distinctive clinical picture and is a chronic hereditary, constitutional disorder. In contradistinction to contact dermatitis vascular-cutaneous hypersensitivity can be demonstrated. A typical case history will tell of the familial occurrence of infantile eczema, asthma, hay fever, and atopic dermatitis. It often starts with a milk crust which changes into infantile eczema, and later on attacks of asthma may alternate with episodes of eczema. Spontaneous improvement occurs in the third decade or later. Atopic derma

weeping. A generalized erythroderma occurs occasionally, cataracts are a rare complication. Personality changes, nervousness and irritability may develop but it is difficult to say whether they are the cause or the consequence of the skin affliction.

If there are only one or a few circumscribed, very pruritic, lichenified patches the eczema is called lichen simplex chronicus or circumscribed neurodermatitis. Common locations are the inner aspects of the thighs, the ankles, and the lateral and nuchal parts of the neck. Lichen simplex is probably a localized form of atopic dermatitis, transitions from localized to disseminated forms occur.

At times distinction between a chronic lichenified contact dermatitis and atopic dermatitis is difficult. In atopic dermatitis we find immediate positive reactions to various allergens such as pollen, dust, and animal protein. Some authors consider a constitutional dysfunction of the autonomous nervous system. Psychologic factors should also be considered since a change of surroundings or climate may have a striking and decisive effect on this affliction.

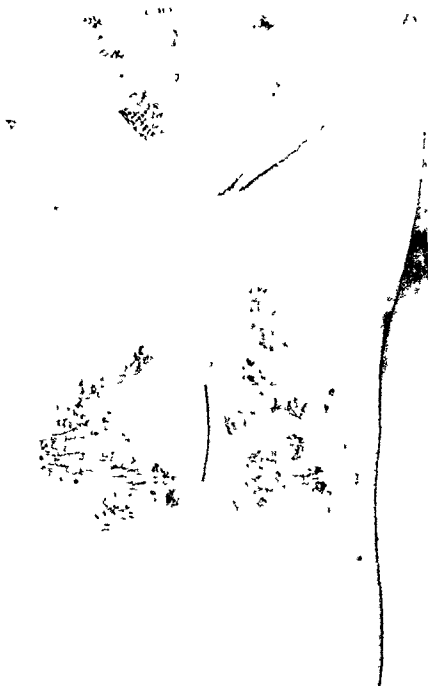




Fig 94 *Atopic dermatitis (neurodermatitis) of neck and face with lichenification and excoriations*

Fig 95 *Patch of lichenified eczema* →



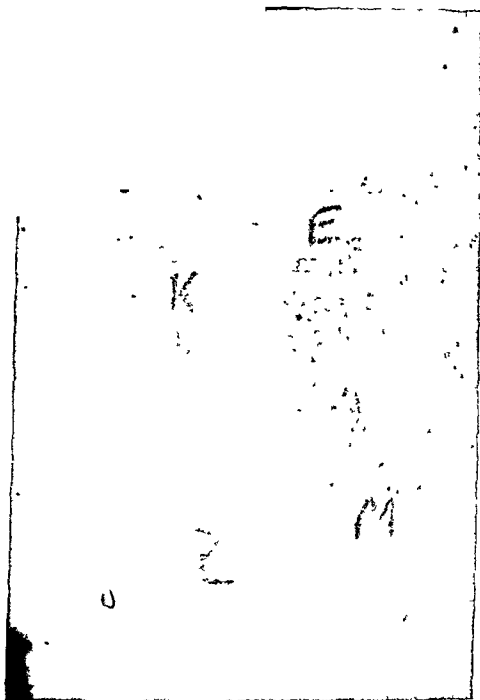


Fig 96 *Positive immediate test to egg white in a baker with atopic dermatitis*
There was a whealing reaction from the scratch test with egg white (*E*) but negative reactions to flour (*M*) sugar (*Z*) and control (*A*)

Infantile Eczema (Fig 97)

Early in infancy groups of papules appear on the face, trunk and extremities, and scales and crusts are present on the scalp (milk crust). Episodes of eczema of varying severity continue for months and years.

Etiologically infantile eczema of this type is a form of atopic dermatitis. It tests with inhalant and food tests. The personal and family history is suggestive of atopic dermatitis, bronchial asthma and allergic rhinitis. It is hardly possible to distinguish the so called true eczema from seborrheic dermatitis of Moro's type. A special entity is Leiner's erythroderma, which is considered a generalized seborrheic dermatitis. Eczema in infants is rarely a contact dermatitis.

Therapy of Eczemas

External Treatment

Acute episodes of eczema often tend to clear up spontaneously. This applies especially to acute contact dermatitis, which generally subsides within a few days after the causative agent has been removed. Therefore, acute eczemas are treated first with mild applications such as wet dressings with saline solution, aluminum hydroxide (Burow's solution, Domeboro), Alibour's solution and with mild ointments containing 3 per cent Burow's solution in cold cream, yellow petrolatum, or zinc oxide ointment.*

When the inflammation is more acute it is advisable to use first an oily lotion such as calamine liniment (U S P) or a "zinc oil" of the following composition (an antibacterial drug is added in case of secondary infection)

Rx	Hexachlorophene, 10 per cent in corn oil (Ar-Ex Cosmetics)	6 0
	(or Zephiran concentrate, 2 4)	
	Ichthyol	1 2
	Zinc oxide	20 0
	Corn oil q s	60 0 cc
	(G-11 Ichthyol zinc oil)	

Where these lotions are too drying they may be alternated with one of the mild ointments mentioned above.

In case of secondary infection wet compresses with potassium permanganate, 1:6000 to 1:9000 are used or Alibour's solution.

* For acute contact dermatitis Dr P A J Smith finds nothing as good as compresses of hydrocortisone lotion 1:1000.

<i>Rx</i> Zinc sulfate	35 0
Copper sulfate	10 0
Camphor water, <i>q s</i>	1000 0

Dilute five to six times for compresses

Mild ointments or pastes for treatment of the subacute eczemas are 2 per cent Vioform or 1 per cent Rivanol in Vaseline, or in equal parts of Vaseline and lanolin or in zinc oxide ointment, Sterosan ointment and paste, or an Ichthyol paste of the following composition

<i>Rx</i> Ichthyol	0 6
Corn oil	3 0
Zinc oxide ointment, <i>q s</i>	30 0

Naphthalan and similar preparations such as Dernaftan are more potent than ichthyol and are well suited for the subacute stage

<i>Rx</i> Naphthalan or Dernaftan	1 5 to 3 0
Zinc oxide ointment, <i>q s</i>	30 0

Antiseptics such as 2 per cent Vioform, 1 per cent Rivanol, or 1 per cent hexachlorophene may be added to these prescriptions

In the subacute and chronic stage, besides wet compresses and ointments tinctures and shake lotions are used

<i>Rx</i> Anthrarobin	3 0
Ichthyol	6 0
Glycerine	12 0
Ether and spiritus dil, <i>aa, q s</i>	120 0
(Arning's tincture)	

Shake lotions are especially useful in widespread eczematous eruptions and occasionally in the acute stages. Calamine lotion is frequently used. A white shake lotion with more 'body' is the following

<i>Rx</i> Zinc oxide	
Talcum	15 0 to 20 0
Glycerine and distilled water, <i>aa q s</i>	90 0

This lotion is more or less creamy according to the quantity of solids which are incorporated. Antieczematous drugs may be added in the form of 2 per cent Vioform, 2 per cent Ichthyol, or 1 per cent hexachlorophene. Alternation of dry treatment in the form of a tincture or a shake lotion with ointments often is helpful. The different medications may be used on the same day, or the dry treatment may be applied for 3 days and then ointments for 1 or 2 days.

A nearly specific agent for eczemas, especially lichenified types, is coal tar. It is usually incorporated in a strength of 3 to 5 per cent in zinc oxide ointment or

yellow petrolatum, but it also can be added in the same percentage to a shake lotion. Crude coal tar may be used undiluted in severe lichenified eczemas, and especially in infantile eczema. After the tar has been applied it is covered with talcum powder, gauze and a bandage. This application can be left in place for 1 to 4 days. After several applications the tar is removed with a mild ointment. Where the color of tar is objectionable, a coal tar distillate such as liquor carbonis detergens, 3 to 5 per cent, in Vaseline or zinc oxide ointment often works nearly as well and is less messy. There are available many commercial, relatively clean tar preparations, some combined with other drugs, for instance Pragmatar (sulfur and salicylic acid), Tarquinor (Quinolol) and Cor-Tar-Quin (Quinolol and hydrocortisone).

Hydrocortisone ointments and lotions have become a most valuable addition to the treatment of eczemas, especially in the chronic stages, but usually not in acute eczemas (for exception see footnote page 133). Where secondary infection exists they should be combined with an antibiotic. Many such combinations are on the market, for example, Neo Cortef, Hydroderm, or Neo-Cort-Dome, all of them containing neomycin as the antibiotic, and Terra-Cortril, which contains Terramycin. Erythromycin ointment may be combined with a hydrocortisone preparation or Sterosan. Hydrocortisone ointment or Vioform-Hydrocortisone cream or lotion may be used. These preparations are especially suitable for lichen simplex chronicus, atopic dermatitis, and eczemas of the ears and anogenital region. Most of these preparations are well tolerated but one must watch for sensitivity to Vioform and neomycin. Antihistaminic ointments are clean and at times are helpful in localized subacute and mild eczemas but they are sensitizers when used topically, *Perazil cream* (Histamin cream in Britain) less than others.

For seborrheic dermatitis sulfur and resorcin are the drugs of choice.

Rx	Precipitated sulfur	15
	Resorcin	15
	Salicylic acid	15
	Yellow petrolatum q s	300

For the hyperkeratotic eczema of palms and soles the following ointment may be used full strength or diluted with equal parts of yellow petrolatum.

Rx	Salicylic acid	30
	Unguentum diachylon, q s	600

In widespread dermatitis short baths often relieve discomfort. One-half cup of soda or oatmeal most convenient in the form of Aveeno colloidal oatmeal, may be added to the tub. In chronic eczemas tar may be added to the bath, commercial preparations are Ar Ex Tar Bath and Zetar emulsion. However, many patients with atopic eczema have a dry skin and should not bathe too

often Drying of the skin should be prevented by use of a bland skin lotion In these cases synthetic vitamin A, 50,000 units twice a day, often is helpful

Internal Medication

Severe attacks of eczema with general discomfort can be shortened by treatment with corticosteroids and ACTH. Prednisolone, at present the favorite, is started at a dose of 5 to 10 mg four times daily for 3 days, then the dose is reduced. As a rule steroid therapy should not be continued for more than 2 weeks. For more prolonged treatment combination with injections of ACTH is recommended. The effect of steroid therapy usually is prompt, often dramatic. In chronic eczemas however, especially in atopic dermatitis there may occur a severe aggravation after the treatment with corticosteroids has been stopped. These drugs are no substitute for proper management of these eczemas. As a rule the adrenal steroids and ACTH should be used only to tide the patient over an acute, distressing phase of dermatitis. In many instances they can be avoided by proper local treatment as described above and by adequate oral doses of antihistamines, with or without additional intravenous injections of these drugs. The dosage for intravenous Benadryl is 30 to 50 mg for Pyn benzamine, 25 to 50 mg, and for Chlor-Trimeton, 2 to 4 mg, once or twice a day. The antihistamines relieve the pruritus but do not influence the eczematous process. Intravenous injection of 10 cc of 10 per cent calcium gluconate or of Calcebronat (Sandoz), once or twice daily, may reduce the inflammation and pruritus. All intravenous injections must be given very slowly. Vitamin B complex orally or by injection, also injections of crude liver extract, appear helpful in some severe cases of eczema. Treatment with arsenic (see "Lichen Planus", page 99) now is only rarely used in chronic eczemas.

In dermatitis complicated by secondary infection, or in those chronic disseminated eczemas in which staphylococci or streptococci possibly play a role, antibiotics may help. Penicillin injections (600 000 units daily), Penicillin V (125 to 250 mg four times a day by mouth, one of the broad spectrum antibiotics such as Aureomycin, Terramycin-SF, Achromycin-SF) or sulfonamides may be given for 4 to 5 days (see page 58).

General Measures

There is no special diet for patients with eczemas. Eczemas provoked by specific foods are relatively rare. A "starvation" diet for instance, of potatoes and tea, or milk and water, or water, orange juice and crackers, may act favorably. Such strict diets should be used for short periods only. It is often advisable to eliminate chocolate, nuts, spices, sea food, coffee, Coca Cola and similar soft drinks and beer. Sometimes a restricted diet with little meat, eggs and fats but plenty of vegetables and fruit is recommended.



Fig 97 Infantile eczema (atopic dermatitis)

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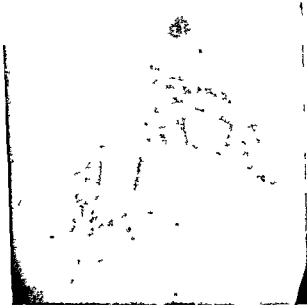


Fig 99 *Urticaria photogenica* The reaction on the right side was produced by irradiation with a mercury arc lamp the wheal and erythema on the left side by exposure to sunlight

Fig 100 *Purpura*
Petechiae and small
purpuric lesions on
the lower leg





A change of environment such as a stay in the hospital or a change of climate often are helpful especially in patients with atopic dermatitis. The beneficial effect may be brought about by rest or by the removal of psychologic conflicts or environmental allergens.

In infantile eczema food allergy plays a much greater role. Foods to which the infant or child is sensitive should be eliminated, but one must take care that the baby is adequately fed. Milk and wheat are the most important food allergens in infantile eczema and can easily be eliminated. Soybean preparations such as Sobee or Mul Soy or a meat base formula adequately replace milk.

In general environmental allergens such as wool feathers or house dust should be eliminated or exposure to them should be reduced. Desensitization to environmental allergens especially house dust is reserved for the most resistant cases of atopic dermatitis and should be started with very weak dilutions for example 1:1 billion.

X Ray Therapy

The course of subacute and chronic eczemas can be shortened by x ray treatments. They should be given very sparingly and only by those who have had experience. 30 to 90 r of a superficial radiation are repeated at 4 to 10 day intervals depending on the size of the dose. After three to six irradiations there should be a rest period of several weeks. It is recommended that x rays be used judiciously and that a total dose of 1000 r per field should not be exceeded during the patient's lifetime.

Urticaria (Hives) (Figs 98 and 99)

The lesion of urticaria is a wheal or hive. It is produced by edema which compresses the blood vessels and causes the white color of the center.

Sometimes they are large and confluent producing the so-called giant urticaria. At times edema of deeper structures occurs and may cause swelling of the lips or eyelids. This is called angioneurotic edema and may be dangerous when it involves the larynx.

The wheals of urticaria develop quickly within 20 to 30 minutes. The individual lesions usually disappear after a few hours without leaving pigmentation or scaling.

Urticaria

Urticaria can be caused by many factors. In other instances there exists an allergy to a certain food or drug.

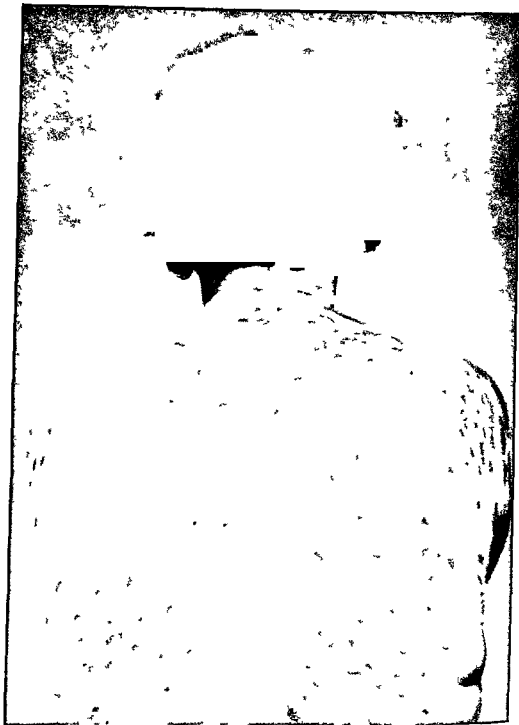


Fig 101 *Drug eruption* The extensive maculopapular drug eruption occurred 20 minutes after the ingestion of a headache pill (Saridon) at the same time the thrombocytes were reduced by 30 per cent The eruption was followed by formation of blisters and scaling and cleared up slowly over a period of several weeks

attacks of urticaria or angioneurotic edema or when antihistamines fail, subcutaneous injections of adrenaline, 0.2 to 0.3 cc are recommended. This dose may be repeated if necessary once, or twice after 15 to 30 minutes. In chronic urticaria antihistamines often are required for a long time, Propadrine hydrochloride, $\frac{3}{8}$ to $\frac{3}{4}$ grain three or four times a day, may help when antihistamines alone are not sufficient, it also counteracts the drowsiness caused by some antihistamines. All these drugs, however, provide only symptomatic therapy.

Causative factors may be sought and if possible eliminated. Desensitization is rarely used in urticaria except in specific instances such as sensitivity to insulin. In chronic urticaria one may try a variety of treatments varying from change of diet to removal of focal infections, antibiotics, intestinal detoxicants such as Sulfasuxidine and sulfaguanidine, or nonspecific therapy including fever or autohemotherapy. Psychotherapy is indicated in certain cases.

In very severe acute cases corticosteroids and ACTH give prompt relief. In urticaria from penicillin penicillinase (Neutrapen) often seems effective.

Urticaria Pigmentosa

Urticaria pigmentosa consists of brownish spots, lentil to coin sized and occasionally much larger, which are transformed by scratching and stroking into wheals. Histologically there is an infiltrate consisting of lymphocytes, leukocytes and mast cells. It is a rare disease of unknown origin, usually occurring in children but occasionally seen in adults. Although it is almost always a harmless disease that is confined to the skin, clearing up spontaneously after many years, very rarely systemic lesions do occur. There is no effective therapy.

Purpura (Fig. 100)

Purpura consists of small red spots produced by capillary hemorrhages. Purpuric eruptions usually are located on the lower extremities and in extensive cases lesions occur on other parts of the body. Involvement of the mucous membranes of the nose, the mouth, the genitals and the intestines may lead to severe hemorrhage. The cause of purpura may be toxic or allergic damage of the capillaries; in some cases a thrombocytopenia is found. The mechanism of blood coagulation usually is not disturbed in contrast to the hemorrhagic diatheses which produce subcutaneous hemorrhages following mild trauma. Purpura may occur as a disease entity or as a symptom of other dermatoses such as eczemas, drug eruptions or microbids.

Thrombocytopenic Purpura

When the number of thrombocytes is reduced capillary bleeding easily ensues. Allergic reactions to drugs such as barbiturates or arsenicals as well as to

Common food causes of acute urticaria are strawberries, fish seafood nuts wheat and milk, among medications, penicillin, aspirin, animal sera vaccines morphine and barbiturates are common offenders.

In chronic urticaria, which may persist for months and years, it is often difficult or impossible to detect a causative factor. Internal infections, infestation with parasites, psychogenic factors or fatigue may play a part. One must also consider the possibility of a conditioned reflex which can be interrupted only with difficulty, or a dysfunction of the autonomic nervous system.

The history may give important etiologic clues. In urticaria from cold wheals may follow a cold bath or exposure to cold weather, wind and rain. In urticaria factitia or dermographia, wheals are caused by friction from clothing or scratching of the skin, and in photogenic urticaria by exposure to sunlight. In these so called physical allergies wheals can be reproduced simply by application of an ice cube, by writing on the skin with a wooden stick, or by exposure of the skin to the sun or to ultraviolet light. Scratch and intradermal tests with foods and drugs in urticaria are rarely positive, however, in the few instances when positive reactions are observed they are often significant. The best way to detect food and drug allergies is to administer the suspected food or drug when the urticaria has disappeared after a strict elimination diet, or a trial diet of tea and potatoes or milk may be followed for several days. If the urticaria disappears new foods are added one at a time until the urticaria recurs.

Urticaria may be combined with symptoms of hypersensitivity of other organs, for example, nausea or diarrhea, migraine or bronchial asthma.

Treatment Because the wheals in urticaria resemble the hives produced by intracutaneous injections of histamine, it is assumed that the urticarial wheal is rates histamine. Antihistamines ly are very effective. A great ly have a sedative side effect,

especially Benadryl and Phenergan which therefore are most valuable at bed time and at night. Antihistamines which give less sedation are Pyribenzamine, Chlor-Trimeton (Piriton), Neo-Antergan, Histadyl, and Thephorin. Average adult doses for oral antihistamines are as follows: Benadryl, Histadyl, Neo-Antergan, Pyribenzamine or Thephorin, 50 mg four times a day, Perazil (Histantin) 50 mg twice a day, Chlor-Trimeton 4 mg four times a day, or Chlor-Trimeton repeat tablets 8 mg twice a day, Phenergan 12.5 mg two to four times a day. In severe cases these doses usually can be doubled or two different antihistamines may be given simultaneously. For children correspondingly smaller amounts are used. When oral antihistamines fail intravenous injections may be added or substituted in adults. Benadryl 30 to 50 mg, or Chlor-Trimeton, 2 to 5 mg once or twice a day injected very slowly. In acute

attacks of urticaria or angioneurotic edema, or when antihistamines fail, subcutaneous injections of adrenalin, 0.2 to 0.3 cc., are recommended. This dose may be repeated if necessary once, or twice after 15 to 30 minutes. In chronic urticaria antihistamines often are required for a long time. Propadrine hydrochloride, $\frac{3}{8}$ to $\frac{3}{4}$ grain three or four times a day, may help when antihistamines alone are not sufficient, it also counteracts the drowsiness caused by some

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When the number of thrombocytes is reduced capillary bleeding easily ensues. Allergic reactions to drugs such as barbiturates or arsenicals as well as to

infections, may cause thrombocytopenia and purpura. In addition there are cases of purpura with reduced thrombocytes without known cause. Werlhof's morbus maculosus is a thrombocytopenic purpura. Purpura fulminans is a rare severe, often fatal form which may follow serious infectious diseases such as scarlet fever, meningococcic infections, septicemia or streptococcic sore throat. Besides punctate purpuric lesions diffuse subcutaneous hemorrhages and, less frequently, blisters and necrosis occur, the thrombocytes are diminished in number and the clotting time is prolonged. The pathogenesis of this disease resembles the Sanarelli-Schwartzman phenomenon, a reaction which can be produced experimentally in rabbits. A preparatory injection of certain bacterial toxins causes damage to the capillaries and the subsequent injection of the same or another bacterial toxin is followed after several hours by a severe hemorrhagic reaction.

Fragility of the capillaries can be demonstrated by the Rumpel-Leede test, where the cuff of a sphygmomanometer is applied to the upper arm for 10 minutes with a pressure of 20 to 30 mm below the maximal blood pressure. Capillary fragility is demonstrated by the appearance of more than 10 petechiae in 1 sq. inch peripherally to the cuff. In each case of purpura, capillary resistance and thrombocytes must be checked. Drugs and infections must be considered as causative factors.

Purpura without Thrombocytopenia

Nonthrombocytopenic purpura is a harmless but often chronic and recurring disease. Schonlein-Henoch purpura probably is an allergic disease with reduced resistance of the capillaries. Rheumatic purpura of Schonlein is an affection consisting of purpuric spots, often accompanied by small scaling and itching papules of the lower legs and occasionally associated with arthritis. The Rumpel-Leede test is negative, only the capillaries of the lower legs appear damaged. Etiologically foci of chronic infections such as granulomas of the teeth, tonsillitis or arthritis have to be considered. This affection may be considered a purpuric microbid.

A similar but rare form is purpura annularis telangiectodes of Majocchi, characterized by circular patches on the lower legs and thighs with slight atrophy and scaling in the center. In Schamberg's disease and also in pigmented purpuric lichenoid dermatitis of Gougerot and Blum the principal symptom is localized pigmentation of the lower legs due to deposits of hemosiderin.

Treatment of Purpura

Drug and other allergies should be recognized and eliminated, and acute and chronic infection should be eradicated. Capillary resistance may perhaps be

increased by oral and intravenous administration of calcium, also by treatment with vitamins such as ascorbic acid (vitamin C), Rutin (vitamin "P") or Mena-dione (Menaphthone B P) (vitamin K). Antihistamines may be tried where an allergic reaction is suspected.

Drug Eruptions (Dermatitis Medicamentosa) (Fig 101)

Drugs are a frequent cause of skin eruptions. There are many people who routinely take sleeping pills, sedatives, analgesics, laxatives or nutritional supplements. The relationship between the causative drug and the dermatitis is often obvious, but if a large number of drugs have been prescribed at the same time, it may be difficult to decide which one caused the eruption. The possibility of a drug eruption must be considered in any atypical dermatosis because drug eruptions may simulate various skin diseases. At times the skin manifestations are accompanied by symptoms of hypersensitivity of internal organs, for example, bronchial asthma, rhinitis, fever, agranulocytosis or gastrointestinal disorders.

Some drug eruptions occur as an allergic reaction between 7 to 10 days after the drug has been taken or administered by injection, such an incubation period is necessary for the formation of antibodies. When the same drug is taken again the dermatosis appears within a few hours, because antibodies are already present. In eczematous drug eruptions patch tests are positive. In urticarial eruptions immediate whealing reactions may be positive, but this is the exception. In purpuric eruptions a thrombocyte count should be made. Sometimes positive test results are obtained which do not correspond to the clinical picture, for instance, a patch test may be positive in urticarial eruptions. In doubtful cases ingestion or injection of a small amount of the suspected drug may be tried, obviously such a trial should be carried out only with great caution.

Types of Drug Eruptions

Urticarial drug eruptions. Penicillin at present is the most frequent cause of urticarial eruptions, which have been observed in 1 to 2 per cent of cases treated with it. Penicillin allergy is usually transitory and disappears spontaneously after a few months. However, penicillin may also cause severe generalized dermatitis and serious and fatal anaphylactic reactions.

Morbiliiform and scarlatiniform drug eruptions may be caused by barbiturates, aspirin and many other drugs. The so called "ninth day erythema" following treatment with arsphenamine or gold is another example. Eczematous drug eruptions have been discussed in the chapter on contact dermatitis. Erythrodermas may occur following the prolonged use of organic arsenic and gold compounds.

Photoallergic eczemas are due to a particular allergy to the combined effect of drug and light (see page 159), they have been observed with sulfonamides p aminosalicylic acid, derivatives of p aminobenzoic acid, Phenergan and chlorpromazine

Fixed drug eruptions are characterized by sharply circumscribed coin to palm sized plaques, with redness, swelling and often pigmentation, they occur always on the same or similar sites when a certain drug has been taken Barbiturates, coal tar derivatives and phenolphthalein, are the most common causative drugs but fixed eruptions may be produced by almost any drug, penicillin, bismuth, sulfonamides or arsenic preparations Skin tests are usually negative but the thrombocyte test (see page 258) may be positive

Another group of drug eruptions is due to the toxic effects of the drug probably aided by an individually lowered resistance Acne due to chlorine, bromides and iodine belong to this category, as does pigmentation following the use of certain oils and ointments (Riehl's melanosis) The pathogenesis of the rare vegetating bromoderma and iododerma is not clear, these drugs may produce cauliflower-like tumors of the skin and mucous membranes which can be mistaken for skin cancer Bromides and iodine also may produce bullous eruptions, the bullous bromoderma and iododerma Treatment with bismuth or mercury may produce grayish lines on the gums which are due to a deposition of the metallic sulfides formed from the circulating metal and hydrogen sulfide liberated by bacteria in the oral cavity The massive use of silver containing medications in former years occasionally lead to a grayish discoloration of the skin and mucous membrane, this argyria was due to a deposition of silver Intensive use of gold preparations at times was followed by a similar chrysisis

Furthermore, there are drug eruptions which present the characteristics of well defined dermatoses, e g , lichen planus or herpes zoster occurring after the use of arsenicals erythema nodosum after sulfathiazole or pityriasis rosea after other drugs In these instances a "biotropic mechanism" (see page 10) is assumed, it is believed that a latent disease is provoked by the drug

Serum Sickness

Some people develop serum sickness through sensitization to foreign protein especially after administration of tetanus antitoxin in which horse serum is used In mild cases 7 to 11 days after the injection erythema and swelling appear at the site of the injection lasting a few days In severe serum sickness especially following the injection of foreign serum into an already sensitized patient, the manifestations are extensive urticaria associated with malaise and swelling and pains of the joints Therefore, if reinjection of tetanus antitoxin becomes necessary bovine serum instead of horse serum should be used Hor-

mones of animal origin *e.g.* insulin or ACTH or the use of vaccines in cases in which allergy to foreign proteins exists may produce reactions similar to serum sickness

Treatment of Dermatitis Medicamentosa

Prompt elimination of the causative drug is the first step. The patient must be alerted not to take the medication again. Topical therapy is similar to that for dermatitis (pages 133 to 134) and urticaria. Antihistamines often help. Corticosteroids are reserved for the more severe cases and are especially valuable in patients with serious serum sickness. A few specific antidotes exist such as British antilewisites (BAL) for lead and gold poisoning and dermatitis and penicillinase (Neutrapen) for penicillin reactions.

Pruritus

Itching (pruritus) is a symptom of many skin diseases but it also may occur as a disease entity as essential pruritus. Pruritus is especially frequent in parasitic dermatoses such as scabies, pediculosis and insect bites. Eczema, urticaria, atopic dermatitis, mycosis fungoides and lichen planus are usually accompanied by pruritus. In other dermatoses itching may occur only following fatigue, excitement or sudden changes of temperature or in the warmth of the bed. Imbalance of the autonomic nervous system plays an important role. For instance, pityriasis rosea or psoriasis rarely cause itching in children and young people whereas the same conditions in older, nervous persons are often accompanied by severe pruritus.

In essential pruritus the skin shows only one objective sign, namely excoriations. Etiologically one has to consider diseases of the liver, diabetes, intestinal parasites, leukemias, lymphogranulomatosis (Hodgkin's disease), and nervous and psychologic difficulties.

Senile pruritus probably is caused by dryness of the skin, a hypofunction of internal organs and arteriosclerosis of the brain due to old age. Occasionally essential pruritus has been observed as an allergic reaction to contactants, drugs and foods.

Pruritus ani and pruritus anogenitalis form a special chapter. The ferments and bacterial flora of the stool tend to macerate the anal skin. This is aggravated by insufficient cleansing after bowel movements and prolapse of the mucous membrane from excessive pressure during defecation. Painful and itching fissures may develop. Hemorrhoids may interfere with cleansing of the anal region and lead to circulatory disturbances through venous stasis. Genital pruritus preponderantly affects the female sex. The vaginal discharge macerates the skin and infection with monilia, cocci and bacilli occurs. Focal infection

has to be considered in anogenital pruritus. Contact dermatitis from a medication often is a complication and emotional factors play a part in its pathogenesis.

Treatment of Pruritus

It is most important to combat these local and general disorders which cause the pruritus. For essential pruritus the following is recommended. Externally, preparations containing hydrocortisone with and without antibacterials (see page 135) are used for localized pruritus. Where the steroid creams and lotions fail or are too expensive, or when large areas are involved antipruritic lotions or creams such as $\frac{1}{2}$ per cent menthol, 10 per cent Calmitol in spirit, or in calamine lotion may be used. Antiscabetic ointments like Eurax often relieve pruritus dramatically. Antihistaminic creams are used less and less because of their sensitizing properties but Perazil cream, a rare sensitizer is an exception. The anesthetics of the "caine" type are best avoided because of their sensitizing potential, but chemically different anesthetics such as Tronothane or Quotane are helpful in some instances of localized pruritus. Tar preparations and alcoholic solutions should not be used on mucous membranes or eroded skin since they are painful.

Internally, antihistamines by mouth or by subcutaneous or intravenous injections usually stop the itching promptly (see page 142), in larger amounts most antihistamines have a pronounced sedative effect, especially when given intravenously. Tranquilizers may be tried. Barbiturates and morphine with its derivatives may increase the pruritus, however, Demerol 50 to 100 mg often is very helpful in aborting an itching crisis. In pruritus and any dermatitis or secondary infection must first be treated (see pages 133 to 134). Hydrocortisone creams combined with antibacterials (see page 135) have superseded most older forms of treatment although if the pruritus is persistent, tar ointments and tar solutions (see pages 134 to 135) are indicated. The rectum should be cleansed after a bowel movement with a mild solution and cotton and later on with water and soap. Large external hemorrhoids should be removed. Internal hemorrhoids may be treated by injections. Underlying allergic, psychogenic and infectious factors must be treated.

Prurigo

conditions described are probably variants of atopic dermatitis. The following conditions are recognized



Fig 10 *Prurigo vulgaris* with excoriated papules of the extensor surfaces of the legs

Popular Urticaria (Strophulus, Lichen urticatus)

This condition consists of groups of oval, pinkish and very itchy papules the size of a cherry pit and in the center of which a small blister or pustule often develops. The affection is observed chiefly in children rarely in adults. It is usually an allergic reaction to insect bites and although food allergy is at times considered it is rarely proved.

Prurigo of Hebra

This rare chronic disease is found in adults who suffered from strophulus as infants. Very itchy, pea sized papules or nodules occur on the extensor surfaces of the arms and legs. Secondary infection leads to enlargement of the regional lymph nodes.

Prurigo Vulgaris (Fig 102)

Similar in distribution to Hebra's prurigo, but much milder, the disease may be caused by the same conditions that give rise to essential pruritus e.g. leukemia, lymphoblastomas, and psychologic, metabolic and allergic disturbances. Not infrequently mixed eruptions are seen, consisting in some instances of elements of atopic dermatitis as well as of eczema and prurigo and in other instances of infantile eczema and papular urticaria, and episodes of atopic dermatitis alternate with prurigo-like eruptions. These cases probably should be considered atopic dermatitis (prurigo of Besnier, disseminated neurodermatitis).

Prurigo Nodularis (Hyde)

Prurigo nodularis is a very persistent highly itching eruption of flat papules occurring mostly on the legs. It occurs chiefly in women and is extremely resistant to treatment.

Treatment of prurigo

Symptomatic, external and internal treatment is similar to that of pruritus. In strophulus disinfection of the home with DDT and similar preparations is recommended to get rid of the causative insects. When foods are suspected elimination and trial diets are tried. Where environmental allergens are considered change to a different apartment or to a different climate may be recommended. Psychotherapy is indicated when marked emotional conflicts are present. In very severe cases even electric shock treatment and sleep therapy have been tried. These methods may now be superseded by the use of chlorpromazine and other tranquilizers.

Erythrodermas

In erythroderma the whole skin is red, infiltrated and scaling, it is a serious disease which persists for months and years. Pruritus, insomnia, loss of proteins, decrease of resistance, and secondary infection may lead to cachexia and death. Erythroderma is often a generalized form of some previously localized dermatosis such as contact dermatitis, especially when caused by drugs, seborrheic dermatitis, atopic dermatitis, psoriasis and, rarely, lichen planus. Erythrodermia desquamativa (Leiner's disease) is probably a generalized seborrheic dermatitis of infants, and Ritter's disease (dermatitis exfoliativa) is

idiopathic erythroderma (Wilson-Brocq). The history and previous observations on the same patient often permit a proper diagnosis, sometimes a histologic examination is helpful.

Treatment. Hospitalization is usually necessary. Local treatment follows the principles of therapy of eczema, and secondary infections are treated by internal antibiotics or sulfonamides (see pages 58 to 59). ACTH and cortisone suppress secondary erythrodermas and are especially helpful in the treatment of acute exacerbations. Superficial x-ray therapy aids in diminishing the inflammation and blood transfusions may be helpful.

VI. SKIN DISEASES DUE TO CHEMICAL AND PHYSICAL AGENTS

Alterations of the Skin Caused by Lyes, Acids and Organic Solvents

The skin protects against external irritants. It is exposed to the action of chemicals from the human environment. The most frequent contacts are alkaline substances such as ammonia, soaps, soda, sodium phosphate, cement or lime, next in frequency are contacts with acids and organic solvents such as gasoline, benzene or turpentine. The horny layer is the main line of defense against these attacks. It may be considered a secretion product of living epidermal cells. The horny layer has a pH of about 5.3, maintained by a high buffering capacity, hence the term "acid mantle" of the skin (Marchionini). Buffers are mainly the proteins and amino acids of the horny layer, sweat and sebum and carbon dioxide, which diffuse from the deeper parts of the skin.

The effects of lyes are of the greatest practical importance. Massive contact produces round, red ulcers surrounded by a gray necrotic margin. More frequent and more important than these massive burns are smaller, often only punctate, necroses and erosions, and diffuse peeling of the horny layer. In housewives, masons and mechanics diffuse patchy redness, fissures and a dry scaly dermatitis may occur. Such damage from alkaline substances may lead to contact dermatitis through sensitization to other contactants. Examples are sensitization to turpentine or detergents in housewives and to potassium dichromate in cement workers. Damage from alkaline substances thus paves the way for an occupational eczema. People with a lowered alkali resistance are especially predisposed to injury from lyes and to occupational dermatitis. Methods have been developed which permit measurement of the resistance to lyes and of the neutralizing capacity of the skin against alkalis (see page 256).

Penetration of alkaline solutions into the nail folds of washerwomen may produce paronychia. When these solutions get under the nail plates a semicircular loosening of the distal end occurs, the so called onycholysis semilunaris.

Hydrochloric acid used in soldering and galvanizing produces pustular lesions. Nitric and hydrofluoric acid are notorious for their necrotizing effect.

Organic solvents such as gasoline, benzene and xylol first have a defatting effect upon the skin. Prolonged exposure produces diffuse reddening and scaling and massive contact causes blisters. Turpentine is a very well known sensitizer. The allergic reactions (contact dermatitis) which are caused by various chemicals have been dealt with in the chapter on contact dermatitis.

Treatment and prophylaxis. Chemical burns are treated with antibacterial solutions and ointments similarly to an infected eczema or pyoderma (see pages 59, 133). Damage due to lyes and organic solvents is often caused by too

intensive cleaning of the skin, especially in industries where it is exposed to dirty and messy substances. Protective clothing and gloves are indicated where possible and in their absence the hands should be cleaned with mild neutral detergents, agents or soaps, protective ointments such as Silicote or Kerodex should be applied to prevent damage.

Alterations of the Skin by Mechanical Agents (Fig 103)

Mechanical irritation of the skin by pressure or friction produces thickening of the skin. The horny layer is increased to several times its normal size, the epidermis is widened and the elastic and fibrous tissue of the cutis becomes hypertrophic. It is for this reason that the skin of the soles and palms is thicker than that of the face or the dorsa of the hands. Sites which are especially exposed to pressure or friction develop localized hyperkeratoses called calluses. Examples are corns from pressure and friction from shoes, and occupational calluses characteristic of special trades and occupations such as in the case of milkers, musicians, etc. Stronger mechanical trauma may lead to rupture of the connective tissue of the cutis and contribute to the formation of striae in fat persons and in pregnant women. In certain people burns, cuts or other mechanical traumas give rise to the exaggerated formation of connective tissue in the form of linear or tuberous dermal growths called keloids. The so called spontaneous keloids are supposed to be the consequences of unnoticed microtraumas.

Treatment Painful and disabling calluses such as corns are first softened by the application of salicylic plaster and are afterwards pared down with a knife. Chronic trauma from shoes should be eliminated. Keloids of short duration may respond to x-rays or radium. In cases that have persisted longer, excision or surgical removal of the protruding tissue should be followed immediately by x-rays or radium, excision alone is usually followed by a larger keloid and therefore is not advisable. Trials with injection of hydrocortisone into the keloids have been promising. This method may be combined with radiotherapy.

Alterations of the Skin by X-Ray

Röntgen rays and the gamma rays of radium and other radioactive elements have long been used for medical purposes. Nowadays these radiations are encountered increasingly in and out of the hospital. Doses of less than 400 r do not produce any visible effect. 400 r and 500 r produce a mild erythema which is usually followed by desquamation. In the treatment of tinea capitis caused by fungi of the *Microsporum* species (see page 36) single doses of over 500 r produce an erythema which



Fig 103 *Knuckle pads* Hyperkeratotic nodules over the joints of the fingers occurred in a man who installed flooring and habitually rested the dorsa of the fingers of his left hand on the floor

occurs in a pattern of three distinct waves (Miescher), a mild early erythema occurring between the first and fifth day is followed by two more pronounced late erythemas most marked around the 20th and 50th day after irradiation. Doses of more than 1000 r produce erythema with vesiculation, massive exposure to several thousand Roentgen units may be followed by early necrosis. Injuries may occur months or even years after irradiation therapy, either after a large single dose or from repeated applications of smaller doses when the cumulative limit of tolerance of 1000 r to 2000 r has been exceeded.

The following are symptoms of late radiodermatitis, in the usual order of development: Dry skin due to atrophy of sebaceous and sweat glands; thin slightly scaling epidermis; telangiectases; hyper and hypopigmentation; permanent epilation; sclerosis of the cutis; slowly healing ulcers; precancerous lesions and squamous cell carcinomas.

Radiodermatitis is characterized histologically by changes in the cell nuclei with increased and pathologic mitoses. The superficial, so-called soft x rays which are largely absorbed in the epidermis are followed by pigmentary changes and telangiectases. The more penetrating so-called hard x rays produce sclerotic ulcers and damage to the underlying structures. During the pioneer days of roentgenology late injuries were frequently observed in patients and physicians; now they are less common and are due either to technical errors or occur following administration of extremely high doses needed for the treatment of carcinoma in cases in which the limit of skin tolerance often must be exceeded. The more severe late injuries often can be avoided if such massive treatment is given in fractionated doses.

Skin Diseases Caused by Sunlight

The Physiologic Action of Sunlight

Radiation from sunlight may be divided into four spectra, according to the wavelength. The short wave spectrum, which is absorbed by the epidermis and exerts the strongest biologic action, is called the short radiation of the sun. The long wave spectrum, which penetrates the atmosphere and exerts the strongest biologic action, is called the long wave radiation of the sun. The short radiation of the sun which penetrates the atmosphere is absorbed by the epidermis and exerts the strongest biologic action. The long wave radiation of the sun penetrates the atmosphere and exerts the strongest biologic action. In this way erythema develops several hours after exposure. The same process increases the pigmentation of the basal cells which is the cause of the suntan. Besides this delayed pigmentation a rare immediate pigmentation is produced mainly by the longwave ultraviolet UV A.

which enables a catalyst to transform a colorless pro pigment into melanin. When the erythema fades the horny layer becomes thickened (Miescher) and able to absorb more short ultraviolet light (UV B), increased tolerance is thus acquired.

An important photobiologic effect of short wave ultraviolet is synthesis in the skin of vitamin D from the provitamin. It is probable that the effect of sunlight on the skin produces further biologically important protective substances, an assumption which is supported by the beneficial influence of sunbathing. Substitutes for sunlight are mercury vapor lamps with potent emission lines in the shortwave ultraviolet. The so called Finsen lamps are carbon arc lamps which have a continuous emission spectrum extending from the ultraviolet to the infrared.

Dermatoses Caused by Light

Sunburn (Erythema Solare)

Sunburn is due to an excessive exposure to light ("phototrauma") which causes changes in the skin in the following order: spotty follicular erythema, then diffuse redness and swelling, with longer exposure, vesiculation is followed by erosions and superficial necrosis, there may be fever and symptoms of general dehydration due to loss of fluids. Histologically there is a necrosis of the upper epidermis with loss of staining reactions and an inflammatory response in the cutis. The causative waves are the short ultraviolet (UV B) which may originate from the sun or from an artificial light source. Atomic explosions also may produce severe ultraviolet dermatitis. Prolonged exposure to infrared rays produces a reticular dilation of the dermal blood vessels with subsequent netlike pigmentation.

Treatment. Mild lotions and ointments are used as in an acute dermatitis (see pages 133 to 134). Pain is relieved by internal analgesics. Benzocaine or Nupercaine ointments may help but are best avoided because of the danger of causing sensitivity. Prophylactically gradual exposure to the sun and the use of protective ointments and lotions containing esters of paminobenzoic acid or other ultraviolet absorbing substances are recommended. A simple protective agent against sunlight is yellow petrolatum. Numerous commercial suntan creams and lotions are available.

Photodermatoses Caused by Photodynamic Action

Substances which otherwise are harmless or of low toxicity may become pathogenic by absorption of light in the skin, a process called photosensitization. It is probably due to an oxidation reaction in which the light acts as a catalyst. The following group of diseases is an example of primary phototoxic sensitivity.

Fig 104 *Meadow grass dermatitis*. These lesions were produced experimentally by short application of a leaf of *Poa annua* followed by irradiation with sunlight



Fig 105 *Spring sunlight dermatosis* (*Fruhlingssicht dermatose* Burckhardt). This boy developed erythema nodules and vesicles on his ears following exposure to sunlight in April





Fig 106 *Polymorphous light eruption* Redness swelling papules and eczematous changes occur on areas exposed to sunlight especially the face

In meadow grass dermatitis (*dermatitis bullosa striata pratensis*) (Fig 104) blisters in a linear configuration occur on the exposed parts of the body after sunbathing on grass or working in a garden. This dermatitis is due to contact with certain plants, followed by sunlight irradiation. Plants such as *Pastinaca sativa*, *Heracleum mantegazzianum*, *Ruta graevolens*, *Dictamnus alba*, *Ficus carica* and *Ammi majus* contain furocoumarins as photodynamic substances. To this group of diseases also belongs the so-called Berloque dermatitis, caused by contact with certain perfumes and exposure to sunlight, in which a linear, inflammatory erythema is followed by a very marked pigmentation. A similar photodynamic dermatitis is due to simultaneous exposure to the oil of the rind of Persian lime and sunlight (Same). ✓

Since coal tar contains photodynamic substances, tar workers often suffer from diffuse dermatitis of the exposed skin. Eosin, also a photosensitizing substance, may be the cause of a photodermatitis from lipstick.

Photodermatoses Caused by Photoallergic Action (Fig 81)

Some persons develop an allergic hypersensitivity to the combined action of light and certain drugs which may lead to a photodermatosis. In these instances we are not dealing with a phototoxic effect which can be produced at will in everybody but with an allergic sensitization which occurs only occasionally. Best known examples are the photoallergic eczemas caused by sulfonamides following the ingestion or external application of these drugs; an acute dermatitis may occur on parts of the skin exposed to light (Burekhardt). Some light sensitivity eruptions observed after the use of paminosalicylic acid, paminobenzoic derivatives, acridine dyes, chlorpromazine and Phenergan are probably also based on a photoallergic mechanism. Photoallergic eczemas have been reported after the use of modern detergents which contain fluorescent optical bleaches.

The diagnosis of photodynamic and photoallergic dermatoses can be proved by the use of photo patch tests. The suspected plant or drug is applied to the skin as a patch test and after 4 to 24 hours the skin is irradiated with sunlight or longwave ultraviolet. In case of photo contact sensitivity only the irradiated test site gives a positive result (see page 255).

Treatment. Symptomatic treatment is the same as in acute dermatitis (see pages 133 to 134). Recognition and elimination of the cause is important, as is prolonged protection against light, since traces of the causative substance may persist in the skin for a long time.

Polymorphous Light Dermatoses (Summer Prurigo) Hydroa Vacciniiforme (Figs 105 and 106)

These eruptions usually occur in spring, but in severe cases they may continue throughout the summer until fall. They are located on areas of the skin exposed

to the sun, the ears dorsum of the nose, cheeks neck and jugular triangle dorsa of hands and fingers A milder form is the sunlight dermatosis of spring ("Fruhlingslichtdermatose", Burckhardt), this occurs especially in children as a papular and vesicular eruption of the ears and face, disappearing within a few weeks The histologic picture resembles that of erythema multiforme with a perivascular inflammation of the cutis which may lead to subepidermal vesiculation

More severe and more chronic are those polymorphous light eruptions which resemble erythema multiforme, or lupus erythematosus and especially the eczematous eruption of exposed areas which may persist throughout the summer In "summer prurigo", pruritus is predominant and scratch marks are present The pinhead sized papules appear after a shorter or longer exposure to the sun depending on the severity of the case The most serious disease of this group is hydroa vacciniforme, in which large blisters on the areas exposed to sunlight may lead to ulcers necrosis and atrophy This is a rare disease which occurs in certain families and is often associated with porphyria

The cause of photosensitivity in these cases is not known As porphyrins have sometimes been found it has been supposed that they may be the photosensitizers According to J Meyer a photoallergy against porphyrin exists in some cases of sunlight eruptions Kimmig found in the urine of patients with polymorphous light eruptions a substance which absorbs light of about 5000Å and Wulf claims that many of the patients are photoallergic to this substance Other photodynamic metabolites have been looked for, and the possibility of a photobiotropic action, as in herpes simplex must also be considered Although herpes simplex is a virus disease, it can be provoked by exposure to sunlight Sunlight may also precipitate skin lesions in lupus erythematosus erythema multiforme and occasionally in contact dermatitis psoriasis and lichen planus

Photogenic Urticaria (Urticaria Solaris) (Fig 99)

A rare but characteristic photodermatosis is urticaria solaris, in which wheals develop after exposure to sunlight With excessive exposure diffuse edema of the skin may occur accompanied by shock and collapse

The carcinogenic action of light is greatly increased in xeroderma pigmentosum a rare familial disease (see page 199)

Treatment Avoidance of exposure to sunlight and the use of protective clothing (long sleeves gloves and large hats) are recommended Protective ointments or lotions the so called sun screens or suntan lotions must protect against the rays which cause the photodermatitis The short ultraviolet rays the sunburn rays are absorbed more or less by sun creams and lotions such as 10 to 15 per cent p aminobenzoic acid which is a good protective agent Numerous commercial preparations are readily available but there is none

which protects against the longer ultraviolet light (UV A) that is often responsible for solar urticaria and polymorphous light eruptions. For the longer ultraviolet and visible light, "physical" sun screen lotions or ointments containing titanium dioxide, or 1 per cent Cinnabar, or cosmetics such as Covermark, are necessary. Sometimes the photosensitivity can be reduced by vitamin B, especially niacinamide, 100 mg four times a day. Treatment with crude liver extract at times is helpful, disturbance of liver function has been considered a cause of many light dermatoses.

Modern antimalarial drugs such as Chloroquin, Plaquenil, and Atabrine have been helpful in a number of photosensitivity dermatoses of the polymorphic light eruption type, and also in solar urticaria. If they fail, antihistamines may be tried, together with gradual exposure to sunlight.

VII. HEREDITARY DISEASES OF THE SKIN

Epidermolysis bullosa

Epidermolysis Bullosa Simplex

Bullae appear following minor mechanical pressure or the friction of every day occupations, especially on hands, feet, shoulders and hips. The blisters contain clear fluid and dry up and heal with the formation of crusts and scales but without scars. Histologically there are superficial intraepithelial blisters. This form of epidermolysis bullosa has a dominant inheritance (Siemens)

Dystrophic Epidermolysis Bullosa (Fig 107)

In this form of the disease the blisters are subepidermal, crusting, as they heal scars, small subepidermal cysts and scleroderma-like papules. The nails on the fingers and toes gradually disappear and scarring leads to crippling of the hands and feet. Involvement of the mucous membranes may cause adhesions in the mouth and conjunctivae and narrowing of the esophagus. This severe malady is usually recessive but occasionally dominant, and the degree of severity is variable even in the same family. A rare complication is vegetating granulations.

Fig 107 *Dystrophic epidermolysis bullosa hereditaria*. The blisters are caused by the friction from the shoes. Note the complete disappearance of toenails.



Fig. 108 *Ichthyo*
vulgaris. The skin is c
vered with blackish d
scales. The inguinal r
gion is spared and the
is only minor involv
ment of the face

*Congenital
epile like
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Fig 110 *Congenital ichthyosis form erythrodermia* The whole integument is reddened and scaly the most marked changes occur on the hands

of the denuded blisters accompanied by generalized amyloidosis. In porphyria cutanea tarda (see page 170) secondary blisters develop following mechanical irritation. In pemphigus vulgaris there is a tendency to develop blisters following friction.

Treatment The patient should be protected against mechanical traumas. The blisters are treated with antiseptic lotions and ointments. Persons afflicted with this disease should be informed before marriage about its heredity.

Abnormalities of Cornification

Abnormalities of keratinization occur as familial disorders, with dominant or recessive heredity.

Ichthyosis Vulgaris (Fishskin Disease) (Fig 108)

Ichthyosis appears during the first year of life and is characterized by a dry and scaly skin. Ichthyosis simplex or xeroderma is a mild form, ichthyosis nitida is more pronounced. Ichthyosis nigricans is characterized by blackish scales. The lower legs, the arms and the back are usually involved. On the scalp there is fine scaling and sparse hair. The skin of the antecubital areas, the axillae and the groins is normal. Secretion of sweat and sebum is reduced. Combination of diffuse ichthyosis with follicular hyperkeratosis, so called lichen pilaris, is frequent. However, lichen pilaris may occur as a disease of its own especially on the extensor surfaces of arms and legs. Lichen pilaris with dry, scaly skin is also seen following chronic eczemas or chronic malnutrition, and in atopic dermatitis.

The resistance of the ichthyotic skin against chemical irritation from acids and alkalis is diminished. Therefore patients with ichthyosis are predisposed to dermatitis and secondary eczemas and should avoid occupations in which contact dermatitis is likely to occur.

Treatment Large amounts of vitamin A, 50 000 units three times daily, often have a beneficial effect on ichthyosis but discontinuation of this treatment is followed by recurrence. The dry skin should be regularly and well greased, especially during the winter. External treatment with sodium chloride is effective. The patient may take a bath in a 3 per cent saline solution or apply a 20 per cent sodium chloride solution to the skin.

Rare Diseases of Cornification

Congenital Ichthyosis (Fig 109) An armor like hyperkeratosis which develops during fetal life usually leads to premature birth or an early death (Harlequin fetus). The few who survive have a skin with reptile-like appearance.

Fig 111 *Hyperkeratosis palmaris et plantaris* Diffuse partially spotty papular hyperkeratosis of palms and soles (below)

Darier's Disease (Keratosis Follicularis). Darier's disease consists of grouped hyperkeratotic lesions which form small spherical papules. The mucous membranes may become involved

Treatment All these abnormalities of cornification may benefit temporarily from large amounts of vitamin A.

Von Recklinghausen's Disease (Neurofibromatosis) (Fig 112)

This condition usually begins around puberty with pigmented spots of varying size scattered over the body. Later on neurofibromas smaller than a cherry pit or as large as plums, and larger, may occur. Also vascular and anemic or depigmented nevi, kyphoscoliosis, and psychic disturbances are seen. Heredity is dominant.

Treatment. Surgical removal of neurofibromas when indicated is the only effective therapy.

Adenoma Sebaceum (Pringle's Disease)

Adenoma sebaceum consists of yellowish and red papular nevi on the face, especially the nasolabial folds. It may be associated with tuberous sclerosis of the brain and mental deficiency and is inherited as a dominant trait.

Other Rare Hereditary Diseases of the Skin

There are further rare hereditary diseases of the skin. *Cutis hyperelastica* (Ehlers-Danlos syndrome) is characterized by loose, hyperelastic skin and easily hyperextended joints. In *Osler's disease* (hereditary hemorrhagic teleangiectasis) there are teleangiectases of the skin, the mucous membranes of the nose and mouth, bladder and intestines which have a tendency to hemorrhage. In *cutis verticis gyrata* folding and furrowing of the skin of the scalp and nuchal region occurs in men. In *anhidrotic and hidrotic ectodermal dysplasia*, some appendages of the skin are absent, e.g. sweat glands, sebaceous glands, hair, nails and teeth. In *Werner's* and *Rothmund's* syndromes, scleroderma-like changes of the skin are combined with congenital or juvenile cataract. In *progeria* and *geroderma* early senility of the skin in children is combined with osseous changes and other malformations (Touraine, Franceschetti).

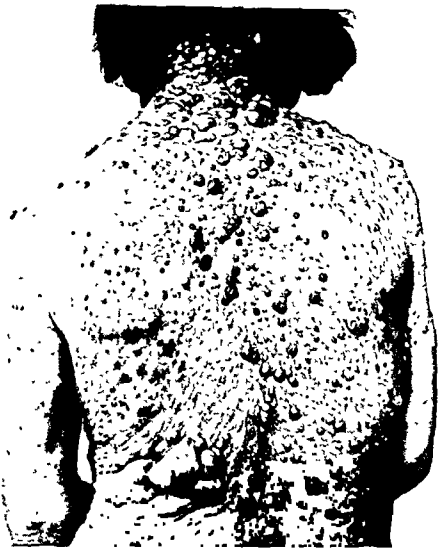


Fig 112 *Neurofibromatosis (von Recklinghausen's disease)* Numerous neurofibromas and pigmented nevi

Congenital Ichthyosiform Erythroderma (Fig 110) In this disease hyperkeratosis is combined with an inflammatory erythroderma, there is also involvement of the antecubital, popliteal and inguinal region which are spared in ordinary ichthyosis

Palmar and Plantar Hyperkeratoses (Fig 111) There may be discrete and punctate lesions or wartlike hyperkeratoses. In the so-called Mal de Meleda, diffuse hyperkeratoses of the palms and soles may be combined with lesions on the ankles and knees

Porokeratosis of Mibelli This extremely rare condition consists of round coin sized hyperkeratoses with a fine, raised, rampart-like edge

page 159) The color of the urine at times is red, and the teeth are pink and fluorescent

Treatment Porphyrria may be helped by a diet protecting the liver and by injections of liver extract and vitamin B complex, as well as by protection against sunlight

Pellagra

Among the various avitaminoses, pellagra produces the most marked changes of the skin. Besides psychic and nervous disturbances and diarrhea, a dermatitis occurs, mostly on areas exposed to light. The involved skin is dry and indurated and shows a yellowish, reddish or brownish color. The eruption is chiefly located on the dorsa of the hands, forearms, face and neck (Casal's necklace), and is accompanied by a burning sensation of palms and soles. Pellagra is due to lack of nicotinic acid and may occur in persons who live exclusively on peeled rice. It may also be caused by diminished absorption of the vitamin in persons with gastrointestinal and liver disorders and also in patients with psychoses who refuse food for prolonged periods.

Treatment consists of large doses of nicotinic acid amide and vitamin B-complex combined with adequate nutrition.

Amyloidosis of the Skin

Lichenoid amyloidosis, a harmless form of amyloidosis of the skin, is a very pruritic papular eruption of the pretibial region of the lower legs which resembles verrucous lichen planus. Amyloid can be demonstrated in histologic sections, or *in vivo* by subcutaneous injections of a 2 per cent solution of Congo red which gives a red color to the papules (Marchionini). Generalized amyloidosis is a serious disorder with swelling and folding of the skin of the face, tongue and lips and involvement of internal organs. A generalized amyloidosis of internal organs may develop from hereditary epidermolysis bullosa (Miescher and Burckhardt).

Xanthomas of the Skin

Xanthelasmas are circumscribed, pinhead to coin sized, yellowish papules of the upper eyelids caused by deposits of lipids, especially cholesterol. They usually appear first on the medial side but gradually the entire lid may be involved. Association with cardiovascular disease is not uncommon. In xanthoma tuberosum, yellowish tumors of the size of a split pea to that of a hazelnut are usually located on the elbows and knees and rarely on other parts of the body, they too are due to a disturbance of the lipid metabolism. Occasionally xanthomas are secondary to hyperlipemia in diabetes. The metabolic disturbance is minor in xanthelasma but marked in xanthoma tuberosum (Schaaf). Granulomas secondarily infiltrated by lipids are discussed on page 104.

Treatment Xanthomas may regress with a diet low in animal fats and cholesterol and high in proteins to protect liver function, alcohol should be avoided. Individual tumors may be excised.

Changes of the Skin in Diabetes Mellitus

Diminished resistance of the skin of diabetics may favor the development of intertrigo, monilia, vaginitis, vulvitis, balanitis and furunculosis. The vascular changes in diabetes may lead to ulcers of the leg, endarteritis obliterans or gangrene of toes. Diabetic tabes, with sexual impotence is a consequence of nerve damage. In all of these conditions the possibility of diabetes must be considered, and repeated sugar examinations in the urine and blood are indicated. Necrobiosis lipoidica diabetorum has been discussed on page 104.

Porphyria

Normally porphyrins are present in the body only in traces, following disturbance of liver function they may be increased and may lead to pathologic changes. Acute porphyria often is provoked by drugs such as barbiturates or sulfones or by lead. It is accompanied by abdominal cramps and psychic disturbances. There are usually no skin lesions. Chronic porphyria (porphyria cutanea tarda) may present bullous eruptions of the hands following trauma or exposure to sunlight and a "sclerodermoid" skin. It is aggravated by drugs and alcohol. Increased amounts of uroporphyrin or its precursors are found in urine and liver. Congenital (erythropoietic) porphyria shows signs of photosensitivity under the picture of hydroa vacciniforme or summer prurigo (see

on the depth of freezing. There are individual differences in the reaction of the vascular system of the skin to cooling. More prolonged restriction of the blood supply is found in acrocyanosis, perniosis (white fingers), Raynaud's disease and sclerodactylia. The degree of the reaction can be measured with the rewarming test (Burckhardt) (see page 257). Malnutrition, insufficient clothing, disturbances of the autonomic nervous system and lowered metabolism are predisposing factors. Women have a greater tendency to abnormal reactions to cold.

Treatment. Therapeutic measures include use of warm clothing and a high caloric diet. Hot beverages, Ronicol (Roche), Priscoline, nicotinic acid and hot baths temporarily improve the circulation. The effect of these therapeutic measures can be gauged with the rewarming test (see page 257). Drug therapy may be combined with local application of mildly antibacterial ointments containing Ichthyol or Vioform.

Fig. 113a. *Pernio*. Red round infiltrates of the dorsa of hands and fingers (Fig. 113a) and of the lower leg (Fig. 113b). The involved skin is slightly cyanotic.



IX. DISTURBANCES OF CIRCULATION AND VASCULAR DISEASES OF THE SKIN

The circulation of the skin fulfills the double purpose of nutrition of the skin and of regulation of body temperature. In cold weather there may be a conflict of these two functions, to prevent too much general cooling the temperature of the peripheral regions may be reduced to such a degree that the nutrition and resistance is inadequate. Circulatory disturbances may be divided into functional changes and organic vascular diseases. Actually a combination of functional and organic disorders is probably the rule because functional disturbances may lead to organic changes.

Vascular Diseases Involving Primarily the Arteries

"White Fingers", Acrocyanosis, Raynaud's Disease

In certain persons minor cooling causes spasm of the digital arteries so that the fingers turn white and cold ("white fingers"). In acrocyanosis the hands, ears, nose and lower legs are cool and bluish red. When it is very cold the skin turns bright red, heat leads to a slight edema. The bluish color of the acrocyanotic skin disappears under compression and when the pressure of the finger is released the bluish color returns from the periphery of the compressed area. The coolness of the skin is due to poor arterial circulation from vascular spasm. The bluish discoloration is caused by dilation of the venous part of the capillaries due to increased venous pressure. Localized acrocyanosis of the legs often is observed during puberty in girls (erythrocyanosis crurum puellarum). In Raynaud's disease there are recurrent attacks of painful arterial spasms which cause blanching with subsequent cyanosis of the fingers. The repeated circulatory insults cause necrobiotic processes of the fingers, with gradual loss of the terminal phalanges, formation of fistulas on the fingertips, and paronychias. Atrophy of the epidermis and sclerosis of the cutis forms a transition to sclerodactylia (see page 107).

Pernio (Chilblain): Injuries Due to Cold (Fig. 113)

Most often involved are the hands and feet and less frequently the nose, cheeks and ears. Burning hyperemic sometimes blistered areas follow prolonged cooling of the skin and may be combined with acrocyanosis. Chilblains occur in wet, cold weather especially at the beginning of the cold season. Real freezing of the skin (frostbite) is due to the effect of low external temperatures and severe cooling through cold winds. To begin with the tissue is stony hard and white, and after thawing it turns bright red, blisters or necrosis occur, depending

on the depth of freezing. There are individual differences in the reaction of the vascular system of the skin to cooling. More prolonged restriction of the blood supply is found in acrocyanosis, pernio, "white fingers", Raynaud's disease and sclerodactylia. The degree of the reaction can be measured with the rewarming test (Burckhardt) (see page 257). Malnutrition, insufficient clothing, disturbances of the autonomic nervous system and lowered metabolism are predisposing factors. Women have a greater tendency to abnormal reactions to cold.

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Fig. 113a. Pernio. Red, round infiltrates of the dorsa of hands and fingers (Fig. 113a) and of the lower leg (Fig. 113b). The involved skin is slightly cyanotic.



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Vascular Diseases Involving Primarily the Arteries

"White Fingers", Acrocyanosis, Raynaud's Disease

In certain persons minor cooling causes spasm of the digital arteries so that the fingers turn white and cold ("white fingers"). In acrocyanosis the hands, ears, nose and lower legs are cool and bluish red. When it is very cold the skin turns bright red, heat leads to a slight edema. The bluish color of the acrocyanotic skin disappears under compression and when the pressure of the finger is released the bluish color returns from the periphery of the compressed area. The coolness of the skin is due to poor arterial circulation from vascular spasm. The bluish discoloration is caused by dilation of the venous part of the capillaries due to increased venous pressure. Localized acrocyanosis of the legs often is observed during puberty in girls (erythrocyanosis crurum puellarum). In Raynaud's disease there are recurrent attacks of painful arterial spasms which cause blanching with subsequent cyanosis of the fingers. The repeated circulatory insults cause necrobiotic processes of the fingers with gradual loss of the terminal phalanges, formation of fistulas on the fingertips, and paronychia. Atrophy of the epidermis and sclerosis of the cutis forms a transition to sclerodactylia (see page 107).

Pernio (Chilblain) Injuries Due to Cold (Fig. 113)

Most often involved are the hands and feet and less frequently the nose, cheeks and ears. Burning hyperemic, sometimes blistered areas follow prolonged cooling of the skin and may be combined with acrocyanosis. Chilblains occur in wet, cold weather especially at the beginning of the cold season. Real freezing of the skin (frostbite) is due to the effect of low external temperatures and severe cooling through cold winds. To begin with the tissue is stony hard and white, and after thawing it turns bright red, blisters or necrosis occur, depending

10 mm in diameter. They are due to an inflammation of the cartilage and the overlying skin, probably caused by a local circulatory disturbance.

Treatment consists of excision.

Vascular Diseases Involving Primarily the Veins

Thrombosis and Thrombophlebitis

Thrombosis of the large pelvic veins and the deeper veins of the extremities may follow severe general diseases or surgical procedures, their discussion is beyond the scope of this book.

Thrombosis and thrombophlebitis of the superficial veins of the skin are milder diseases and occur mostly in varicosities of the lower legs. Around the involved vein which usually cannot be felt in the acute stage, is a painful infiltrated plaque. The temperature is increased and the blood sedimentation rate is elevated.

A special form is thrombophlebitis migrans and saltans. Smaller, superficial veins and their branches are indurated and are sensitive to touch. The overlying skin shows a linear redness. This type of phlebitis may occur *simultaneously or successively* on various parts of the body such as the legs, abdomen or arms.

Treatment. As this form of thrombophlebitis is supposedly based on infection, penicillin and other antibiotics are indicated (see pages 58 to 59). Bed rest is recommended during the acute phase but later on restricted ambulation is permitted. The lower legs and sometimes the thighs as well should be bandaged. In severe cases anticoagulants such as heparin, Dicumerol or Tromexan are given. When the last two drugs are used dosage must be regulated by Quick's prothrombin test so that the prothrombin time is reduced to between 30 and 20 per cent. Butazolidin tablets have also been recommended.

Venous Stasis and Its Complications (Figs. 114 and 115)

Stasis dermatitis and stasis ulcers may occur as a consequence of varicosities but may also be due to previous thrombophlebitis and thrombosis of the deeper veins of the legs, with subsequent *permanent* occlusion of the veins. The development of varicosities is favored by occupations that require constant standing, tight clothing that obstructs circulation and by repeated pregnancies. Often impairment of the circulation caused by varicosities and thrombosis becomes manifest only when the general circulation fails, as in cardiac decompensation. The symptoms of venous stasis are varices of the thighs and lower legs, edema of the lower legs and feet appearing at the end of the day, sclerosis of the cutis, atrophy of the epidermis above the ankles and isolated purpuric and hyperpigmented spots on the lower legs. The most significant complications are ulcers and eczemas of the lower legs. The latter are due partially to the disturbed



Endarteritis Obliterans (Buerger's Disease)

Endarteritis obliterans of the legs with intermittent claudication is an organic arterial disease affecting mainly the muscles, only rarely do ulcerations on the legs and toes occur in its final stages. The temperature of the skin is reduced and an arteriogram discloses narrowing of the femoral artery. Endarteritis of the legs often is only part of a general inflammatory disease of the arteries which involves the heart, intestines and brain.

Periarteritis Nodosa (Kussmaul)

Periarteritis nodosa is a rare inflammatory disease with subcutaneous, often hemorrhagic nodules of the skin. Besides the severe form milder manifestations occur, with subcutaneous nodules of the lower legs which resemble erythema induratum. These skin lesions respond to chemotherapy and antibiotics.

Chondrodermatitis Nodularis Chronica Helicis (Winkler)

Painful, hard, small nodules appear on the rims of the ears, especially of men who often have been exposed to cold weather. The lesions are about 3 to

Treatment of the varicose veins is important in the management of stasis ulcer and dermatitis but it is best to wait until the ulcer has healed, or at least until the infection has completely subsided. The decision as to whether varicose veins should be treated by ligation and/or surgical stripping, by excision, or by injection of sclerosing agents, rests with the surgeon. At present surgical removal is preferred, but sometimes ligation combined with retrograde injection of the veins is performed. A thorough evaluation of the problem is necessary because not all patients benefit from surgical intervention, in some instances, especially with severe tissue damage, the patient's condition may become aggravated. Special care must be taken where there is a history of previous thrombophlebitis or thrombosis of the deeper veins for in these cases the superficial dilated veins may help to carry the blood and sclerosing them can further impair the venous return. Perthes' test gives information about the retrograde circulation of the deeper veins. The test is carried out as follows. A pressure bandage is applied below the knee, so that it compresses the superficial veins. The patient first is allowed to walk around, then he lies down and the leg is elevated. If the varicose veins have been filled, and fail to empty after elevation of the legs, this means that the deep veins are not patent and injection of the varicose veins is contraindicated. Modern sclerosing agents are fatty acid derivatives such as sodium morrhuate and Sylnasol, or wetting agents such as sodium Sotradecol, 0.5 to 1.0 cc of the solution is administered by injection, with the patient in a sitting position and an elastic bandage is applied. In further treatments the amount and concentration of the medication is adjusted according to the degree of reaction following the first injection.

Diseases of the Lymph Vessels

Lymphangitis and Lymphadenitis

Inflammation of lymph vessels and lymph nodes occurs in numerous infectious skin diseases such as *pyoderma*, *erysipelas*, venereal diseases and virus infections. Squamous cell carcinomas and melanomas may invade the lymph nodes.

Elephantiasis

Severe and repeated inflammation of the lymph vessels may lead to their destruction. Consequently, lymph stasis develops which eventually may lead to massive edema of the skin and underlying tissue, causing a condition called elephantiasis. Tropical elephantiasis usually is due to destruction of the lymph passages following infection with *Filaria bancrofti*. Elephantiasis nostras occurs after repeated attacks of *erysipelas*, when legs, arms and genitals may become

circulation and partially to bacteria, and not infrequently maintained or aggravated by sensitization to local medication. Occasionally the ulcers are very small, only the size of a pinhead, and very painful, these are due to thrombosis of minute blood vessels. Usually they are larger, sometimes involving a greater part of the lower legs, they often are multiple and heal with scarring. The arterial circulation may also be disturbed. Most leg ulcers are stasis ulcers but many other conditions may cause ulcerations on the legs. One must consider ecthyma, tertiary lues, diabetes, tuberculosis (such as ulcerated erythema induratum), periarteritis nodosa, sickle cell anemia, and at times squamous cell carcinoma.

Treatment The principles of the treatment of stasis ulcers and stasis eczemas are elimination of the infection and improvement of the circulation. If the infection is only mild and superficial, local antiseptics and antibiotics such as Vioform, Rivanol or Ichthyol, either as an ointment or paste, or shake lotions (see pages 133 to 134) are sufficient. If there is a more severe or resistant infection internal antibiotics or sulfonamides are used. In recurrent and persistent cases underlying factors such as focal infection, diabetes, anemia or malnutrition must be looked for and corrected. Generous amounts of vitamins, minerals, proteins and other essential foodstuffs may be helpful.

Patients with stasis ulcers or stasis dermatitis seem more prone to develop hypersensitivity to topical medications. Therefore, before trying a new medication it is advisable to test it with a patch test. Potent sensitizers such as the sulfonamides, penicillin, Furacin or ammoniated mercury are best avoided in these conditions. Dried red blood cells, marketed as Lyocyte are very helpful after the acute irritation has subsided. It is necessary to fill the whole ulcer with a generous amount of the powder, the bandage usually can be kept in place for 2 or more days. This is also an ideal dressing for the ulcer prior to the application of the Unna's paste boot (see below).

The circulation is improved by elevating the legs as much as possible by raising the foot end of the bed about 6 inches, resting the legs on a footstool while sitting, and standing for only short periods. In ambulatory patients a piece of sponge rubber may be placed over the ulcer after it has been dressed with the appropriate medication and an elastic bandage or an elastic stocking applied afterwards. Drugs which produce local vasodilation, e.g., Priscoline (25 mg four times a day) or Roniacol (25 mg four times a day or more) appear helpful. Patients with badly infected, long lasting ulcers are best hospitalized. After the infection has cleared up but before the ulcer has healed, a supportive light cast, such as Unna's paste boot is applied to be left in place for 2 or 3 months. In case of pain, discomfort or excessive draining the boot should be removed immediately.

Fig 114 *Stasis dermatitis and stasis ulcer* Varicose veins of the lower leg and sclerosis of the cutis with atrophy of the epidermis and pigmentation in the lower third of the lower leg. An ulcer has developed in the center of the sclerotic area.



Fig 115 *Stasis ulcer* with dermatitis of the surrounding skin.

several times their normal size. Localized vesicular dilation of lymph vessels occurs, they may burst and lymph may extravasate. Lymphogranuloma venereum (Nicolas Favre) not uncommonly leads to elephantiasis of the penis, scrotum or vulva. Surgical removal of lymph nodes in cases of cancer also may be followed by elephantiasis like swelling.



Fig 117 *Senile keratoses* Circumscribed lesions with a whitish adherent hyperkeratosis on an erythematous base



Fig 118 *Senile keratosis* The horny layer is much thicker than normal and nuclei are visible (parakeratosis). The epidermis is thinned and the papillae are flattened. There is an irregular arrangement of the cells of the basal layer. Some nuclei are darkly stained and lobulated, some contain two nuclei. In the cutis there is an infiltrate of lymphocytes with occasional leukocytes.

Fig 116 *Subcutaneous fibromas* Subcutaneous fibromatous tumors overlying the finger joints and attached to the tendons



X. BENIGN TUMORS

Nevi or Moles

The designation "nevus" covers a group of congenital, often tumor-like, malformations of the skin

Pigmented Nevi

Pigmented nevi are small tumors, papular or flat, light or dark brown. The size varies greatly. Histologically there are hyperpigmentation of the epidermis and clusters of nevus cells often surrounded by nerve fibers in the papillae and cutis. Everybody has some pigmented nevi which usually present only a cosmetic problem. Degeneration into a malignant melanoma generally is rare but may occur when pigmented nevi are exposed to constant irritation or trauma as on the sole of the foot.

Special forms are verrucous nevi and hairy nevi (nevus pilosus), nevi spili present only hyperpigmentation without nevus cells. The "Tierfell" nevi of the German literature are darkly pigmented and very hairy nevi which involve larger, often segmental, areas of the skin and resemble animal fur.

Blue nevi are small, firm, dark blue nodules. Microscopically spindle shaped, pigment forming cells are found in the cutis.

Vascular Nevi (Hemangiomas)

Nevus flammeus (port-wine stain) or nevus teleangiectaticus is a congenital, sharply circumscribed discoloration of the skin. The color at birth is bright red, but later in life changes into a violet blue as the blood vessels gradually become more dilated (G. Miescher, U. W. Schnyder). The redness disappears under pressure. Microscopically there is only dilation of blood vessels but no tumor like proliferation.

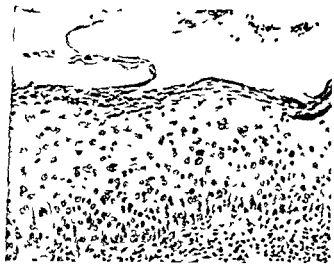
There are two groups of nevus flammeus. One is usually symmetric and located mainly on the nape of the neck, forehead, eyelids, and the sacral region. They are found at birth in 47 per cent of the newborn and mostly disappear spontaneously within a few years, as they are found in only 19 per cent of adults. They require no treatment.

The second group of nevus flammei is mostly unilateral but at times also symmetric and is frequently associated with other malformations. Combinations of nevus flammeus with hypertrophy of the subcutaneous tissues and bones are recognized as special entities. In the Klippel-Trenaunay syndrome the leg is usually slightly longer and varices are present. The Parkes-Weber syndrome presents in addition arteriovenous anastomoses and the Sturge-Weber-Krabbe syndrome glaucoma and angiomatosis of the meninges.



Fig 119 *Bowen's disease* This is a papular lesion covered with massive yellowish scales. After their removal an infiltrated erythematous easily bleeding base becomes visible.

Fig 120 *Bowen's disease* The epidermis is widened and there are no papillae visible. Within the epidermis there are numerous multinucleated cells with clumped nuclei. Several mitoses are visible. There is no stratum granulosum. The horny layer contains many nuclei. In the cutis there is a dense infiltrate of round cells which have partially migrated into the epidermis.



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Tuberous hemangiomas (strawberry marks) are more or less prominent bright red or bluish red tumors. Histologically they consist of increased blood vessels with active proliferation. Because of the large, cavernous, hollow spaces they are also called cavernous hemangiomas. They appear after birth, usually within the first few weeks of life, and within the first few months they increase rapidly in size. Many disappear or regress spontaneously during the first 5 or 6 years of life.

Other forms of vascular nevi are nevi aranei, or spider nevi, they consist of starlike or spider-like dilations of small vessels which originate from a small central blood vessel. Senile hemangiomas are pinhead sized to lentil sized bright red or bluish papules, which may occur in large numbers in older people, they consist of cavernous dilations of small blood vessels.

Rarer forms are the lymphangiomas, with blister-like, dilated and increased lymph vessels. They occur especially on the lips, tongue and chest. Sebaceous nevi occur as yellowish verrucous plaques on the scalp. Comedo nevus consists of a localized accumulation of comedo-like structures.

Treatment The ideal treatment for pigmented nevi is surgical excision or complete destruction with electrocautery. For cosmetic reasons partial removal often is preferred and thus the protruding part of the nevus is removed with scissors or a knife and the base is treated with superficial fulguration. The macular hemangiomas (nevus flammeus) are very difficult therapeutic problems. They do not respond to regular x-ray therapy. Electrodesiccation, application of solid carbon dioxide, and intensive ultraviolet irradiations with a Kromayer mercury arc lamp not infrequently are followed by ugly scars and pigmentation. Plastic surgery may produce good results. Where such a procedure is not feasible or desired, thorium X lacquer or Grenz rays may be tried, these often give satisfactory results but rarely lead to complete disappearance. Cavernous hemangiomas usually respond well to x-ray and radium treatment. Small cavernous hemangiomas may be treated with carbon dioxide snow or destroyed by electrocoagulation. Nevi aranei are treated with electrolysis or electrocoagulation.

Fibromas (Fig. 116)

Hard fibromas occur as reddish nodules the size of a cherry pit. Soft fibromas occur in the form of pedunculated tumors. Fibrosarcoma protuberans will be discussed with the sarcomas. A special form is the subungual, sometimes multiple fibroma (Polland). Neurofibromas are discussed as von Recklinghausen's disease (see page 169). Knuckle pads are subcutaneous fibromatous tumors connected with the tendons overlying the interphalangeal joints.

Treatment Excision is the only treatment for fibromas.

Other Benign Tumors

Epithelial cysts, sebaceous cysts and lipomas are usually discussed in surgical texts. Among other rare benign skin tumors the following may be mentioned here. Leiomyomas are cherry pit sized, painful, brownish-red, small tumors, usually occurring in groups. Histologically they consist of smooth muscle fibers. Histiocytomas are solitary, firm nodules. Microscopically there is an increase of histiocytes, and spindle shaped cells are arranged in intertwining and anastomosing bands. Endometriomas are nodules in the region of the buttocks and thighs of women, they enlarge during menstruation. They represent a misplacement of uterine mucosa into the skin. Syringomas are lentil sized, brownish tumors usually occurring in large numbers, microscopically they are cysts originating from the sweat glands.

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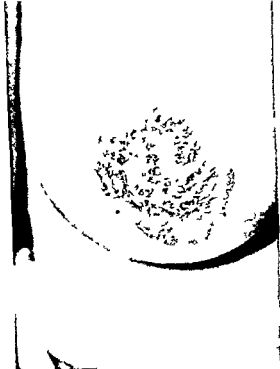


Fig 121a *Paget's disease of the nipples* Sharply circumscribed eczema like plaque with redness infiltration and scaling covering the nipple and surrounding area this condition had slowly developed over a period of years

Fig 121b *Paget's disease* The epidermis is irregularly widened and covered with a parakeratotic horny layer which contains darkly staining clumped nuclei. In the epidermis there are Paget cells with round partially kidney shaped and clumped nuclei rich in chromatin and a light protoplasm. There is a plasma cell infiltrate in the cutis.



XI. PRECANCEROSIS OF THE SKIN

Precanceroses are conditions which after several months or years may turn into malignant tumors. Some are more likely to do so than others. Accordingly one may distinguish between precanceroses in a strict sense (conditions that usually or often turn into malignancy) and precanceroses in a wider sense (conditions with an increased tendency to malignant degeneration). Among the obligate precanceroses are senile keratosis, cornu cutaneum, Bowen's disease, Paget's disease, the melanotic precancerosis (melanotic freckle), verrucous leukoplakia, hyperkeratosis caused by tar or arsenic, and the verrucous lesions of xeroderma pigmentosum. Precanceroses in a wider sense are the atrophic changes of the skin following x-ray, radium or extensive ultraviolet irradiation, lupus vulgaris, lupus erythematosus and the flat, smooth leukoplakia. Precanceroses in the widest sense are ordinary pigmented nevi, scars and chronic ulcers.

Senile Keratosis (Figs 117 and 118)

Senile keratoses are the most frequent precanceroses. They are located on areas exposed to sunlight, *i e*, the face, dorsa of hands, rims of ears and the bald scalp. They originate as circumscribed, brownish or reddish spots with small hyperkeratoses which later on become diffuse. Formation of crusts and an inflammatory halo may follow. These keratoses develop over a period of months or years and occur, singly or in large numbers, in persons beyond 50 years of age. A special form is the cutaneous horn (cornu cutaneum) with horn like hyperkeratosis. Histologically hyperkeratosis and parakeratosis are found also narrowing of the epidermis and loss of papillae. The nuclei of the epidermal cells may be irregular, polymorphic and rich in chromatin. There are increased mitoses, multinuclear cells, and a slight inflammatory infiltrate in the cutis (Fig 118).

Bowen's Disease (Figs 119 and 120)

This relatively rare form of precancerosis with very scaly, psoriasis like coin sized papules is mainly located on the face, trunk and genital area, but also on arms and legs. On the glans penis, prepuce and vulva it occurs in the form of sharply circumscribed erythematous lesions which are called erythroplasia of Queyrat. They develop slowly over a period of months and years and almost always degenerate into a squamous cell carcinoma. Metastasis to lymph nodes and internal organs occurs. Histologically the epithelium is thickened, mitoses are more pronounced than in senile keratosis. Large clumped nuclei (clumped cells) are characteristic. There is spotty intracellular edema (Fig 120).

Treatment Radium or x-rays are well suited for the treatment of senile keratoses and Bowen's disease. Single irradiation with 1000 to 1500 r at 32 to 70 kv. (half value layer 0.2 to 0.8 mm aluminum), or with Grenz rays at 12 kv., 1500 r three or four times, may be used. Curettage and electrodesiccation, followed by radium treatment in the case of Bowen's disease, is very satisfactory. Surgery is a very good method when complete excision is feasible. Benign senile hyperkeratoses, especially when multiple, may be treated with liquid nitrogen or wire brush surgery.

Paget's Disease (Fig. 121)

Paget's disease develops on the nipple and the areola of the female breast, rarely in men, and only occasionally on other locations such as the genitals. There is a sharply defined redness with weeping and scaling which clinically may resemble a parasitic (microbic) eczema. One must become suspicious of Paget's disease when an eczema-like condition of the nipples progresses slowly and does not respond to antieczematous treatment. Paget's disease in most instances is combined with a cancer of the ducts of the mammary gland. Histologically the epidermis is widened and contains nests of large, edematous cells with nuclei rich in chromatin; these so-called Paget cells are separated from the surrounding cells because of the disappearance of the intercellular bridges. The cutis shows a plasma cell infiltrate.

Treatment Amputation of the breast is the method of choice, combined, when indicated, with the removal of the regional lymph nodes and postoperative x-ray therapy.

Melanotic Precanceroses (Lentigo Maligna, Melanotic Freckle)

Figs. 122 and 123)

Lentigo maligna was described by Dubreuilh and Hutchinson. It consists of a sharply defined pigmented spot of irregular size, the color varies from dark brown to black. The lesion grows slowly and as time goes on prominent or infiltrating nodes occur (malignant melanoma). Histologically there is hyperpigmentation and nests of segregating cells with irregular, darker staining nuclei, partially dropping into the dermis.

Treatment In the smooth, precancerous stage, surgical excision or electrocoagulation is adequate treatment, when there is a suspicion that a malignant melanoma has developed. Wide surgical excision or x-ray therapy are indicated.

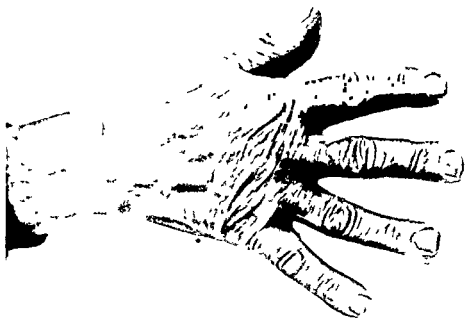


Fig 122 *Melanotic precancerosis (lentigo maligna)* Irregular, sharply demarcated black spots on the dorsal aspect of the third and fourth finger

Fig 123 *Melanotic precancerosis* In the center of the picture there is visible a nest of cells with irregular nuclei which penetrates into the cutis



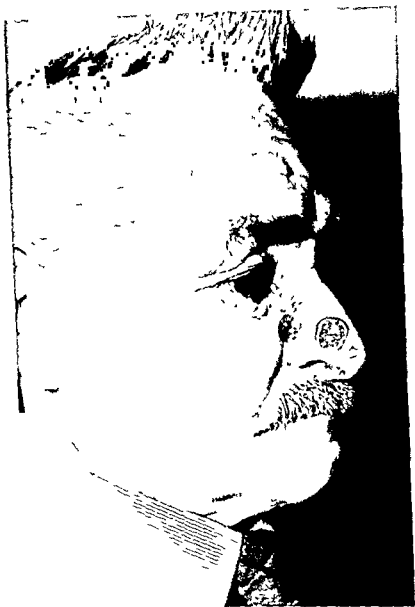


Fig 124 Basal cell carcinoma Ulcers with a firm wall like elevated edge Slow progress during the course of many years

XII. MALIGNANT TUMORS OF THE SKIN

Carcinoma of the Skin

Basal Cell Carcinoma (Rodent Ulcer) (Figs 124 to 128)

A basal cell carcinoma (basal cell epithelioma) may develop from a senile keratosis or more often directly from apparently normal skin. In a precancerous lesion the base under the crust becomes uneven and somewhat infiltrated, and friable granulations are formed. When the basal cell epithelioma develops from normal skin there are first pinhead sized, pearly lesions and later coin sized whitish papules with a central depression which often turn into an ulcer. The elevated edge slowly progresses peripherally and the central ulcer may heal with a scar. Histologically there are nests of cells without intercellular bridges which have darkly staining, sometimes irregular, nuclei and show increased mitoses. The proliferating cells originate from the basal layer and strands of cancer cells penetrate into the dermis.

Without treatment a basal cell carcinoma may invade deeper structures and destroy the skin, the underlying subcutaneous tissue and even the bones and blood vessels (carcinoma basocellulare terebrans). Basal cell carcinomas do not produce metastases, but there may be a transition into a mixed baso-squamous epithelioma with metastases.

A special form is the superficial basal cell carcinoma (multiple, pigmented pagetoid basalioma, Arning's carcinoid), which usually presents numerous lesions on the face, trunk and extremities. These lesions superficially resemble an eczema but have pigmented, pearly nodules on the periphery and a slightly red scar in the center. Some of these tumors, over a period of several years, may turn into penetrating, destructive carcinomas. Other rare forms are Brooke's epithelioma adenoides cysticum, and cylindroma, with prominent hazelnut sized tumors on the scalp, back of neck, face and back. Histologically they are relatively benign basal cell epitheliomas with central degeneration and formation of cysts.

Squamous Cell Carcinoma (Prickle Cell Carcinoma) (Figs 129 to 131)

Squamous cell carcinoma often originates from a precancerosis such as a senile keratosis or a cutaneous horn. Carcinomas of the lips, penis, vulva and anus, as well as tar and x-ray cancers, are usually squamous cell carcinomas. Within a few weeks firm, prominent nodules the size of cherry pits or hazelnuts develop, they soon break down or form ulcers with a firm base and a wall like



Fig 126 *Pigmented pagetoid basal cell carcinoma* Lesions in the temple region are groups of pinhead sized mostly black pigmented nodules The tumor progresses peripherally while the center heals and forms a depigmented and hairless scar

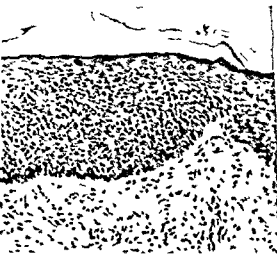


Fig 127 *Basal cell carcinoma* A cluster of epithelial cells grows down into the cutis from a thin epidermis The cells within the tumor mass are irregular The nuclei are partially darkly stained partially light and often have an irregular structure Multinucleated cells and occasional pathologic mitoses are found



← Fig 128 *Pigmented superficial basal cell carcinoma*
Lesions on the trunk are sharply outlined. Redness is visible, as are pinhead sized nodules especially at the edge. Some of the nodules show brownish blackish or bluish pigmentation.



Fig 129 *Squamous cell carcinoma* → of the lower lip



Fig 130 *Squamous cell carcinoma*. Strands of carcinomatous cells grow into the cutis from a thickened epidermis. Partial keratinization in the center of these strands and occasional mitoses are visible.





Fig 132a *Malignant melanoma with metastases in the lymph nodes*
A small bluish black nodule developed within a few weeks into a cherry sized blackish tumor with massive regional metastases (Courtesy of Prof G Miescher Dermatological Clinic Zürich)

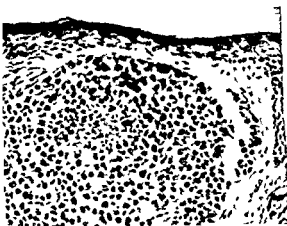


Fig 132b *Malignant melanoma*
Beneath the thin epidermis is an accumulation of cells with round darkly staining nuclei partially with irregular structures. There is no connecting stroma between the cells ("bag of potatoes") (Courtesy of Prof G Miescher Dermatological Clinic Zürich)

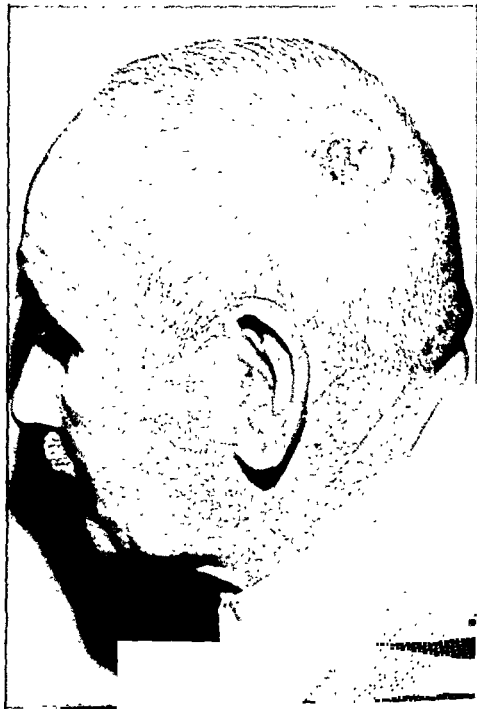


Fig 131. *Squamous cell carcinoma of scalp.* Nodular tumor with central ulceration
Rapid growth within a few weeks

edge After several weeks to months, metastases may occur in the regional lymph nodes Without treatment they may develop into fist sized necrotic tumors or palm sized vegetating plaques

Histologically there are strands of proliferating epithelial cells penetrating into the dermis They possess intercellular bridges and there is a definite tendency for keratinization and formation of horny pearls The nuclei of the cells are rich in chromatin, with increased, often pathologic, mitoses Multinucleated cells may be found and also an irregular, whorl like arrangement of cells

In recent years a benign, probably infectious tumor of the skin has been recognized which clinically and histologically greatly resembles squamous cell carcinoma Keratoacanthoma also called mollusum sebaceum and pseudo-carcinoma, occurs as single or multiple tumors on the face and hands and in rare cases on other parts of the body Hazelnut sized hard, red tumors develop within 1 to 3 months, with central necrosis and ulceration Spontaneous cure occurs after several weeks or months Only clinical experience and observation permits distinction of these tumors from carcinomas

Treatment The preferred treatment of skin cancer is radiotherapy with x-ray or radium Superficial x-rays are usually given in divided doses, depending on the size of the tumor For all small and large superficial epitheliomas, curettage and electrodesiccation followed by irradiation with heavily filtered radium is a very satisfactory method Large, penetrating skin cancers require deep x-ray therapy in fractionated doses At times radiotherapy and electrosurgery may be combined Surgical and electrosurgical excision, with or without subsequent plastic procedures, may produce equally good results, but usually is more complicated In carcinomas that are radio resistant and in other recurrent carcinomas of the skin, Mohs' chemosurgery produces excellent results The prognosis is generally good with basal cell epitheliomas, in squamous cell carcinomas it depends on early treatment Regular check-ups are advisable to detect local recurrences or metastases in squamous cell epitheliomas and the development of skin cancers in other locations

Etiology and Prophylaxis A number of factors are known which favor the development of precanceroses and carcinomas of the skin The location on the exposed parts of the body points to the significance of sunlight The tendency to develop sunlight induced cancers is especially great in the familial xeroderma pigmentosum in which skin cancers arise in childhood on parts of the body exposed to sunlight The carcinogenic effect of ultraviolet has been proved by animal experiments in mice Late radiodermatitis due to excessive exposure to x rays or gamma rays from radium or other radioactive elements may lead to the development of carcinoma Coal tar is known as a chemical carcinogenic agent, causing cancer of the scrotum in chimney sweeps In mice, repeated

Fig 133 *Lymphatic leukemia* Massive symmetric tumorlike infiltrates of the skin form the so-called leonine facies



Leukemia of the Skin (Fig 133)

Skin lesions in leukemia are considered metastases of the disease. They are more frequent in lymphatic leukemia less in the myelogenous form. The following manifestations are distinguished:

1 Prurigo with very pruritic papules or small infiltrates. Microscopically they present the characteristic cellular elements of lymphatic or myelogenous leukemia.

2 Erythroderma of an eczema like character with diffuse redness, infiltration and scaling of large parts of the skin.

3 Tumors, often massive and of a bright red color. These are often located symmetrically on the face and lobes of the ears (leonine facies). Microscopically there is a massive infiltrate of lymphocytes (Fig 133).

Kaposi's Idiopathic Hemorrhagic Sarcoma (Kaposi's Sarcoma)

This is a rare disease which begins with hazelnut to cherry sized bright red or bluish red tumors usually on the feet and lower legs. They are surrounded by hemorrhagic red and yellow discoloration and pigmentation of the skin. Over a period of years there is a gradual extension producing in addition infiltrated hemorrhagic lesions on the trunk and the extremities. After many years the internal organs also may be affected by similar tumors. The outcome is usually fatal. Microscopically the tumors consist of newly formed blood vessels and infiltrates composed of round and spindle shaped cells.

Treatment: X-ray therapy and penicillin may produce temporary improvement.

Hodgkin's Disease

This is a malignant disease of the lymphatic system which occasionally appears first on the skin. Nonspecific skin manifestations are pruritus, dermatitis and pyodermas. Specific for the disease are nodular infiltrates and tumors with a typical granulation tissue presenting the Sternberg Reed giant cells. The diagnosis is facilitated by enlarged lymph nodes and splenomegaly which usually accompany the disease.

Treatment is similar to that in mycosis fungoides but nitrogen mustard and triethylene melamine (TEM) seem to be more effective in Hodgkin's disease.

Mycosis Fungoides

This is a rare dermatosis. The first manifestation, as a rule, is the eczematoid so-called premycotic stage with diffuse redness and scaling of the skin and usually severe itching. After several years patchy infiltrates occur and some turn

applications of coal tar or its main carcinogenic constituent benzopyrene produce cancer within a few weeks Scars especially following burns and chronic inflammatory ulcerations may degenerate into cancer Prophylactic measures include avoidance of too prolonged exposure to sunlight and of soiling with tar Regular observation of older people for precancerous lesions and small epitheliomas as well as early treatment can prevent the development of incurable skin cancers

Melanoma (Figs 132a and b)

A malignant melanoma may develop from normal skin from a lentigo maligna or from a pigmented nevus They have been called by Miescher "melanomalignomas" The first sign is a cherry sized dark black or bluish black tumor, usually combined with a blackish or bluish discoloration of the surrounding skin The bluish color cannot be removed by pressure because it is due to pigmentation and not to a dilatation of blood vessels Without treatment the tumors may reach the size of a plum or a fist In the so called amelanotic melanoma pigmentation is nearly absent, the bright red vegetating tumors easily may be mistaken for a granuloma pyogenicum Malignant melanomas often metastasize early into lymph nodes bones, lungs and skin

Histologically these tumors consist of masses of roundish or spindle shaped cells with numerous mitoses, the nuclei are rich in chromatin The cells have no connection with each other and resemble a bag of potatoes Since some believe that a biopsy may provoke metastases, it seems more cautious to precede the biopsy with x ray treatment in case of suspicion of a malignant melanoma

Treatment Early radical surgical excision or x ray therapy of the primary tumor may be curative Where metastases are present the prognosis is very poor

Sarcoma (Dermatofibrosarcoma Protuberans)

Sarcomas of the skin are rare Fibrosarcomas consist of cherry sized firm bright red tumors which occur singly or in groups and grow slowly Excision is often followed by local recurrences The tumors are radio resistant and metastases occur only rarely Therapeutically electrosurgical excision is recommended

Skin Metastases of Malignancies Arising Elsewhere in the Body

Skin metastases of carcinomas or sarcomas of other organs or from other skin regions are called secondary malignancies of the skin Skin metastases of cancer of the breast occur with relative frequency either as single coin sized tumors or as a diffuse infiltrated carcinoma the so called cancer en cuirasse

XIII. DISEASES OF THE APPENDAGES OF THE SKIN

Diseases of the Sebaceous Glands

Sebaceous glands are distributed all over the skin but are especially plentiful on the face, scalp, chest and back. Sebum lubricates the horny layer and regulates the wettability of the skin. It also has a disinfecting and neutralizing function. (Dunbar, Miescher) The secretion of sebum develops during puberty and decreases with old age. There is more secretion in summer than in the cold winter, hence the dry, chapped skin in winter time.

Acne Vulgaris (Figs 134 and 135)

Simultaneously with an increased secretion of sebum a disturbance of keratinization stops the discharge of the sebum. The latter is oxidized in the excretory ducts of sebaceous glands and turns dark. It is in this way that the comedos, the primary lesions of acne vulgaris, originate. They can be expressed with a comedo squeezer. Secondary inflammation due to infection leads to inflammatory papules, pustules and nodules, producing pustular and nodose acne. Occasionally extensive subcutaneous infiltrates are formed with cutaneous or subcutaneous abscesses, so called acne conglobata. Acne is located primarily on the face, back of the neck, back and chest and occasionally also on the upper arms and thighs. It may be accompanied by visibly increased secretion of sebum (seborrhea) and a seborrheic dermatitis. Acne occurs during puberty and in adult life. Occasionally a few comedos and acne pustules are present in nearly everybody.

Treatment of Acne Vulgaris

Therapy of acne consists of peeling and antiseptic applications as well as internal medications and general measures.

External medication. Most of the external medications used have a peeling and antiseptic effect. The most common are salicylic acid preparations, which are used less frequently. Other preparations are resorcin and sulfur preparations, although these are not as effective. Such as the following:

Rx 3 per cent salicylic acid 6 per cent colloidal sulfur, in Neobase or a similar base

A stronger and greasier ointment contains

Rx 5 per cent salicylic acid 5 per cent resorcin and 5 per cent precipitated sulfur in yellow petrolatum

into mushroom like solid tumors which later ulcerate. Occasionally the disease begins as a tumor, the so called mycosis fungoides d'emblee. Internal organs also may become involved. After many years death ensues.

Histologically the tumors consist of a granulation tissue which contains large histiocytes the mycosis fungoides cells.

Treatment No cure is known but health and life can be prolonged for several years. X ray therapy is probably the most efficient therapeutic weapon. Arsenic urethan, nitrogen mustard, cortisone, adrenocorticotropin (ACTH) and antibiotics have also been used.

1 yr
12 yr
myc

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Treatment of Acne Vulgaris

Therapy of acne consists of peeling and antiseptic applications as well as internal medications and general measures.

External medication. Most of the external medications used have a peeling and antiseptic effect. The main ingredients are salicylic acid, sulfur and resorcin and less frequently antiseptics and estrogens. Lotions are usually preferable to ointments although in some cases of dry skin it is well to alternate with a cream, such as the following:

Rx 3 per cent salicylic acid 6 per cent colloidal sulfur, in Neobase or a similar base

A stronger and greasier ointment contains

Rx 5 per cent salicylic acid, 5 per cent resorcin, and 5 per cent precipitated sulfur in yellow petrolatum

A milder more elegant application is the following alcoholic solution

Rx	Resorcin	3 0 to 6 0
	Salicylic acid	3 0 to 6 0
	Glycerin	12 0
	Spiritus 70 per cent q s	120 0

Commercially prepared lotions are usually preferred because they are more stable of uniform quality and cosmetically more acceptable

Polythionate lotion contains sulfur, Sulforcin lotion sulfur and resorcin Acnestrol sulfur and stilbestrol and Premarin lotion contains estrone sodium sulfate Sulforcin base Acnomel Eskamel and Acnid are pleasant creamy preparations containing both sulfur and resorcin In case of pronounced infection one may alternate with disinfectant ointments such as 3 per cent ammoniated mercury ointment or antibiotic ointments

The addition of hydrocortisone to acne lotions as in Cort Acne lotion or Acne Cort Dome cream at times produces superior results over the plain anti acne preparations

Strongly peeling ointments are rarely used any longer

✓Compresses may be applied with Vlemminckx's solution a time honored effective preparation Under the name of Vlem Dome it is now available in a readymade perfumed powder which somewhat masks the unpleasant odor of the preparation

One must remember that resorcin and especially ammoniated mercury are sensitizers

X ray therapy should be used sparingly and not at all in the pure comedo acne It often is helpful when other methods fail Ultraviolet treatments of the whole body are beneficial and can be carried out at home by means of one of the inexpensive ultraviolet light emitting bulbs (General Electric Westinghouseylvania)

Unightly scars resulting from a bad case of acne now can be satisfactorily improved by the wire brush technique of Kurtin

Internal management of acne Internal and parenteral measures must be adjusted to the particular form of acne and to the patient In severe pustular acne and acne conglobata antibiotics and sulfonamides often are very helpful They may have to be continued for weeks or even months but often a small maintenance dose will suffice Immunization with staphylococcic toxoid or with a mixture of staphylococcic toxoid and a staphylococcic vaccine may be added to the treatment program when antibiotics and sulfonamides do not produce the desired effect Synthetic vitamin A 100 000 units daily given over many months helps in some cases where seborrhea is a marked factor large amounts of

vitamin B and C and at times injections of crude liver extract are beneficial. Thyroid properly adjusted to the patient is helpful. natural and synthetic estrogens are beneficial in girls with ovarian dysfunction and in boys with severe pustular and nodular acne. Gonadyl (an oral preparation of serum gonadotropin) is giving promising results.

It is well to place the patient on a diet eliminating chocolate, nuts, condiments and rich foods and reducing the amount of animal fat. The worst offender in acne is chocolate.

Psychologic treatment by the attending physician should be given in every case because many boys and girls with acne suffer from an inferiority complex often far out of proportion to the disfigurement caused by the disease.

Industrial Acne, Acne Medicamentosa (Fig. 136)

Certain chemicals used in industry may produce comedos, papules and pustules which resemble acne. In machine workers oil may be splashed on the hands and arms and may produce oil acne. Perchlornaphthalene is an insulating material with a characteristic odor widely used in the electrical industry. If there is insufficient ventilation it may produce acne of the face (perna disease). Coal tar occasionally causes tar acne. Prolonged treatment with bromides as in epileptics regularly produces bromide acne as does intensive treatment with iodine. Inunction of ointments containing irritating hydrocarbon compounds also may produce an acne like eruption.

Treatment consists of elimination of the causative factor and the same external treatment as in acne vulgaris.

Acne Rosacea, Rosacea (Fig. 137)

Rosacea occurs in middle aged and older persons and is characterized by pustules, bright red papules and a diffuse slightly infiltrated redness with telangiectases of the face especially the nose, nasolabial folds, forehead and chin. The pores of the skin may be dilated and the sebaceous glands of the nose often increase at times so much that they produce a rhinophyma. Cold and heat as well as improper medications increase the redness. Histologically there is a marked increase of the sebaceous glands and an inflammatory infiltrate in the cutis occasionally of tuberculoid character.

The etiology of the condition is not clear. One has to consider the relation with disturbed liver function, gastric anacidity, emotional factors and even allergies. An infectious origin in the sense of a tuberculoid also has been considered (rosacea like tuberculoid) especially when lupus like nodules with a tuberculoid structure are present.

Treatment Local measures include the application of wet compresses (see page 133) during the acute phase, in the chronic stage, the same topical treatment is used as in acne vulgaris (see page 203) At times 3 per cent ammoniated mercury, 3 per cent Vioform, or antibiotic creams are helpful

Exposure to heat should be avoided. In case of an acidity, Acidulin is recommended Large amounts of vitamin B and C and at times crude liver extract are very helpful When an infectious origin is suspected, a search for focal infection should be instituted and, if found, elimination of the focus, or treatment with antibiotics or sulfonamides may be helpful During the climacteric at times estrogens in women and testosterone in men are indicated

The diet is about the same as in acne vulgaris, but coffee, tea and alcoholic beverages, especially beer, are also forbidden

Rhinophyma is treated surgically

Acne Necrotica (Acne Varioliformis)

This is a relatively rare condition which is not connected with real acne except by its name The condition consists of small pustules which turn into crusted erosions and small ulcers, often leaving punched out scars The main location is the scalp, especially the hairline and temple areas, occasionally it is seen in the bearded region and in other parts of the body Acne varioliformis is persistent and tends to recur The etiology is unknown, but infection with a special strain of *Micrococcus pyogenes* (*Staphylococcus pyogenes*) is suspected

Treatment Acne varioliformis is difficult to treat because it tends to recur Local treatment must be continued for weeks after the lesions have cleared Sulfur and resorcin sulfur ointments, as used in acne vulgaris, and especially antibiotic ointments (see page 58), are helpful Intraderm tyrothricin is a clean medication for the scalp Vitamin A, 100,000 units daily for many months, and immunization with staphylococcic toxoid may be tried in resistant cases

Diseases of the Hair

During the first half of fetal life the skin is covered with lanugo hair, except on palms and soles As a disturbance of development this condition may persist throughout life and give the skin the appearance of pelt (hypertrichosis universalis, "dog face" hypertrichosis) During the second half of fetal life and after birth the firm final hair of the scalp develops, as do the eyebrows, eyelashes and the hairs of the nostrils and external auditory canal During puberty hair grows in the genital area, the axillae and on chest and beard of men The hair covering the body becomes more coarse with age, especially in men In women the hair of the scalp grows faster—between 0.3 and 0.8 mm daily When the maximal



Fig. 134 *Acne vulgaris* with pustules and nodules



Fig 135 *Acne vulgaris* with comedos papules and pustules

Fig 136 *O. facie* with comedos papules and pustules





Fig 137 A ne uro ea w h rh nophyma and pus ules

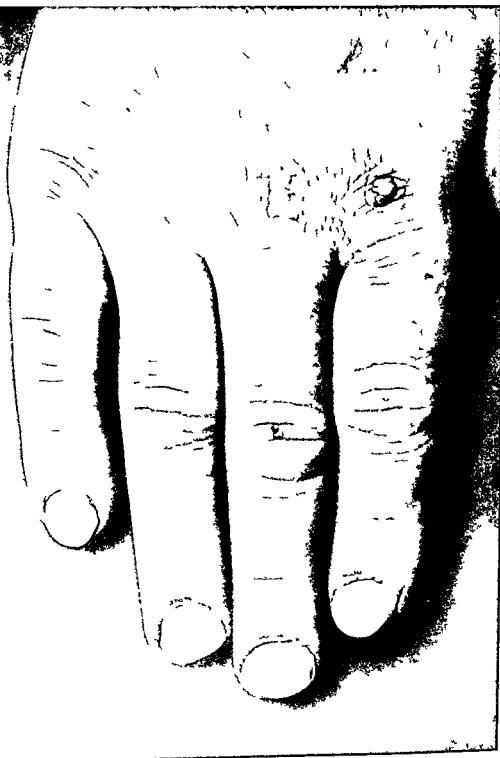


Fig 138 *Koilonychia (spoon nails)*

length has been reached the hair falls out and after a certain resting period the same papilla forms new hair

Hypertrichosis

Hypertrichosis of the female sex is mostly a constitutional affair. Occasionally it is due to an endocrine disturbance such as hermaphroditism or adrenal cortical tumors

Treatment Localized areas of hypertrichosis are best treated with electrolysis or electrodesiccation but in extensive cases shaving is recommended. Patients should be warned against any attempts at x ray epilation. Where endocrine disturbances are present they should be corrected

Seborrheic Alopecia

There is a mild chronic inflammation of the skin of the scalp either diffuse or spotty with increased formation of scales (pityriasis simplex). In severe cases in which scaliness is massive and asbestos like the condition is called *tinea amiantacea*. In seborrheic alopecia the hair becomes sparse but there is no complete baldness

Treatment Selenium sulfite (Selsun) shampoo now has become the treatment of choice in seborrheic alopecia. Antiseborrheic and antiseptic alcoholic lotions and creams also may be used as follows

Rx	Euresol	2 4
	Salicylic acid	2 4
	Topisol	12 0
	Spiritus 70 per cent q s	120 0

A stronger more active preparation is the following

Rx	Liquor carbonis detergens	5 0
	Resorcin	
	Salicylic acid aa	3 0
	Glycerin	6 0
	Castor oil	1 0
	Spiritus of lavender q s	120 0

For antiseborrheic ointments and creams see page 203. Vitamin B-complex plus C and in case of secondary anemia iron are helpful

Senile and Premature Alopecia

As men grow older they develop alopecia beginning in the temple regions and on the vertex which leads to an extensive baldness of the central parts of the scalp. The skin becomes smooth shiny and atrophic. In certain families this

baldness occurs early, between 25 and 35 years, and is called premature alopecia. Its cause is not known. It does not occur in eunuchs and females. However, in women the hair may become thinner after the menopause and may become quite sparse in old age.

Postinfectious and Toxic Alopecia

A diffuse loss of hair may occur 6 to 8 weeks after a serious illness such as pneumonia, typhoid or erysipelas, or after delivery, but the hair grows back when the patient recovers. The same disorder is observed after attacks of severe poisoning, especially with the rat poison, thallium.

Alopecia Areata

Loss of hair occurs in roundish patches which expand peripherally. Clinically there are no signs of inflammation, but microscopically perifollicular round cell infiltrates are found. The hair surrounding the patches can be easily pulled out. In some spots there are stumps where the diameter of the hair is thinner near the opening of the follicle ("exclamation point" hair). There may be only one bald spot, although often they are numerous. Occasionally there is a band like alopecia on the back of the neck or sides of the scalp, called ophryiasis. Regrowth of hair may occur after several months, but the alopecia may spread and involve increasingly larger areas of the scalp and beard. A worse form, alopecia areata maligna, also involves the eyebrows, eyelashes and all hair of the body. In extensive and chronic cases a permanent, spotty or complete baldness may develop. The cause of alopecia areata is not known. Nervous and emotional factors and focal infection have been considered.

Treatment There is no satisfactory treatment. Disinfecting, slightly irritating topical medication may be tried, such as

Rx	Beta-naphthol	.				10
	Salicylic acid	10
	Colloidal sulfur	.			.	20
	Washable cream (Neobase or the like), q s					300
	(Apply twice a day)					

The following is an alcoholic solution

Rx	Tinctura capsica	60
	Resorcin	40
	Salicylic acid	20
	Glycerin	30
	Spiritus, 70 per cent, q s	600

Ultraviolet irradiation either with the mercury arc lamp or the so called cold quartz lamp may be tried likewise a small amount of x ray or thorium X It is by no means certain that local treatment has any beneficial effect besides its psychologic value Cortisone and ACTH in large doses have produced regrowth of hair even in severe cases but this treatment is not recommended since the hair falls out again as soon as the medication is discontinued /

Alopecia areolaris with its small spots of loss of hair is a different disease it is a consequence of a syphilitic exanthema of the scalp

Alopecia Combined with Atrophy of the Skin

Circumscribed roundish hairless spots with atrophic skin are observed after several skin diseases such as lupus erythematosus, lichen planus atrophicans or morphea or in an independent disease called pseudopelade Irregular loss of hair with atrophy and scar formation is seen following favus ulcerating pyoderma burns tuberculosis of the skin and tertiary lues

Diseases of the Shaft of the Hair

Drying of the hair shaft leads to a longitudinal splitting and breaking of the hair (trichoptilosis) or to a nodular splitting of the hair (trichorrhexis nodosa)

A rare familial affliction is monilethrix (beaded hair) with sparse and short hair and spindle shaped swellings Graying of the hair (canities) is a physiologic process of aging In certain families graying begins in youth (canities prematura) Circumscribed graying of the hair is seen in alopecia areata in vitiligo of the scalp and in poliosis

Diseases of the Nails

Dystrophies of Nails

Transverse Lines (Beau's Lines)

During prolonged or severe illness for instance typhoid grippe scarlet fever measles etc there may be a transitory damage of the matrix of the nails with the result that an inferior thin irregular nail plate develops This shows up and advances slowly as a transverse line of the nail The daily growth of the nail is between 0.1 and 0.14 mm The time of the past illness can be estimated by the distance of the transverse line from the nail fold

Onychomycosis

Clawlike growth of the nails may follow trauma of the nail or be due to familial disposition

Treatment Treatment consists of filing off the claws or removal of the diseased nails

Koilonychia (Spoon-Nails) (Fig 138)

In this condition the nails are thin and fragile and concave instead of convex. It has been described in iron deficiency anemias but may occur for no apparent reason.

Onycholysis

There is a loosening of the distal end of the nail from its bed. Often the loose part forms a half circle (onycholysis semilunaris), at times there is a hyperkeratosis of the nail bed. The cause of this disorder is not clear, but frequent contact with alkaline soaps and detergents seems to favor its development.

Inflammatory, Infectious Nail Diseases

Paronychia

Paronychia is an inflammation of the nail fold showing redness, swelling and a purulent secretion. It is caused by pyogenic cocci such as *Staphylococcus* and *Streptococcus*, or by fungi, especially *Candida albicans*, contributing factors are damage from manicure or from alkaline soaps. Occasionally a syphilitic chancre in the nail region may resemble a paronychia with ulceration.

Treatment Pyogenic paronychia is treated with hot soaks of Alibour's solution or potassium permanganate, and with antibacterial ointments (see pages 58, 59), in severe cases internal antibiotics or sulfonamides are indicated. Monilial paronychia responds well to Vioform, Arning's tincture (see page 134) and x-ray treatment. Avoidance of moisture and of damage from alkaline cleansing agents is important, rubber gloves should be worn for dishwashing and laundering.

Mycosis of the Nails

In dermatophytosis and tinea and also in moniliasis, fungi may invade the nail and transform it into a gray brittle mass. Fungi can be demonstrated microscopically in small particles of the nails which may be scraped off with a knife or removed with an electric drill.

Treatment Onychomycosis is often very resistant to treatment especially in cases due to *Trichophyton purpureum*. The diseased parts of the nail should be filed off or drilled away with an electric drill and antimycotic treatment (see dermatomycosis, page 39) should be applied. Removal of the diseased nails is often followed by a recurrence.

Unguis incarnatus (Ingrowing Toenails)

This disorder usually affects the big toe, the lateral parts of the nails grow into the soft tissues, causing fissures, granulomas and small abscesses.

Treatment When trimming of the nail is not sufficient, removal of proud flesh and of the lateral parts of the nail may be necessary. Sometimes excision of the whole nail and/or the lateral part of the matrix of the nail as well is required. Antibacterial ointments are used after treatment. In order to prevent recurrences the patient is advised to wear wide shoes which do not compress the toes and to avoid cutting his nails too short.

Diseases of the Sweat Glands

There are two forms of sweat glands. The eccrine sweat glands occur all over the body and are especially numerous on palms and soles. Their cylindric cells produce a true secretion, slightly acid in character. The large apocrine sweat glands constitute the second form. They occur on the axillae, on the areola of the breasts and in the anogenital region. They produce a slightly alkaline secretion and part of the cellular protoplasm is discharged with it. The main purpose of secretion of eccrine sweat is regulation of the body temperature. The apocrine glands are under the influence of the sex hormones.

A constitutional oligohydrosis or anhidrosis is found in ichthyosis vulgaris, in anhidrotic dysplasia and in old age. Anhidrosis may be dangerous because of overheating of the body.

Hyperhidrosis is found to be most frequent during puberty as a consequence of a disbalance of the autonomous nervous system. It is especially disturbing on palms and soles. Hyperhidrosis of the axillae often is accompanied by an unpleasant odor (bromhidrosis). Emotional stress may increase the secretion of sweat.

Treatment Hyperhidrosis can be treated locally with commercial so called "deodorants". Applications of 10 per cent formalin in alcohol or formalin powder are more potent. Internally one may try Bellergal, anticholinergic drugs such as Banthine, Pro-Banthine and Prantal, also Benadryl or barbiturates. In severe cases which interfere with the patient's ability to work, x-ray treatment may be tried.

Miliaria rubra or prickly heat is an inflammation of the eccrine sweat glands, with numerous reddish papules on the trunk and the extremities. It follows intensive sweating in patients secondary to fever or residence in the tropics.

Hidradenitis axillaris (*hidradenitis suppurativa*) of the apocrine glands has been discussed in the chapter on pyoderma (see page 57).

For Fordyce disease is a rare disorder consisting of very itchy papules in the axilla, mamillary area and groins. It is a chronic inflammation of the skin connected with dysfunction of the apocrine sweat glands.

Koilonychia (Spoon-Nails) (Fig 138)

In this condition the nails are thin and fragile and concave instead of convex. It has been described in iron deficiency anemias but may occur for no apparent reason.

Onycholysis

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Unguis incarnatus (Ingrowing Toenails)

This disorder usually affects the big toe, the lateral parts of the nails grow into the soft tissues causing fissures, granulomas, and small abscesses.

XIV PIGMENTARY DISORDERS OF THE SKIN

The pigment of the skin is formed by the dendritic cells (melanocytes) of the basal layer of the epidermis. The role of the enzyme dopa oxidase, which forms pigment from its precursor dihydroxyphenylalanine can be demonstrated by means of Bloch's dopa reaction. Fitzpatrick considers tyrosine as the first step in the formation of pigment which is converted into dopa by tyrosinase, further oxidation produces melanin.

Complete depigmentation occurs in vitiligo (Fig. 139). This is a disorder of unknown origin characterized usually by symmetric patches of depigmentation. The marginal parts occasionally show hyperpigmentation and the openings of the hair follicles often are the last parts to lose the pigment and also the first ones to show repigmentation in cases of spontaneous cure. The hair in the involved areas is white.

A special form is leukoderma centrifugum (Sutton's disease) where depigmentation occurs around a pigmented nevus. The dopa reaction is negative within the vitiliginous area. A dependable treatment is not known but Oxsoralen (Meladinine) (8-methoxypsoralen) in conjunction with exposure to sunlight is claimed to give satisfactory cosmetic results in approximately 20 to 30 per cent of patients treated for prolonged periods (3 to 9 months).

Permanent or transitory hypopigmentation or depigmentation occurs in a great number of skin diseases in which the pigment producing cells are damaged. Examples are leukoderma following secondary syphilitic eruptions and psoriasis, spotty depigmentation following sunburn, and depigmentation following eczema of the genital or other regions.

Permanent depigmentation occurs as a consequence of the treponemal disease pinta which is common in Mexico and Central America. Hyperpigmentation also may occur after inflammatory dermatoses. Examples are pigmentation following lichen planus, impetigo, pityriasis rosea and photodynamic inflammations due to perfumes (Berloque dermatitis) or to coal tar (Riehl's melanosis).

Ephelides (freckles) are irregularly distributed small pigmented spots which become especially prominent after exposure to sun (Fig. 140). Sunlight produces both darkening and an increase of the pigment (see page 155). An endocrine origin is assumed for the hyperpigmentation of the skin in Addison's disease and also for the patchy symmetric pigmentations in young women, especially during pregnancy (chloasma uterinum). All irregularities of pigment formation are more marked following irradiation with sunlight. Therefore, sun screens are indicated (see page 160). The internal use of 1 to 2 g of vitamin C may cause a

The so called "dyshidrosis" has no relationship to sweat glands. It is an eczematous vesicular and bullous inflammation of the hands and feet referred to as cheiropompholyx and podopompholyx respectively, and is often due to a fungus infection or an eczema. It is debatable whether or not an idiopathic dyshidrosis exists.

Syringomas are tumors of the sweat glands.

transitory regression of hyperpigmentation Benoquin cream (monobenzyl ether of hydroquinone) has been effective in the treatment of hyperpigmentation especially chloasma in about 50 per cent of the cases, but causes contact dermatitis in a considerable number of patients

Incontinentia pigmenti (Sulzberger-Bloch) usually is a congenital disorder

of other organs are often present, especially of the eyes, brain, teeth and hair. The most characteristic manifestation is a bizarre, irregular, slate gray pigmentation with sharp, jagged outlines, it often fades out after several years. The disease occurs predominantly in females. No effective treatment is known.

Pigmentation of the skin may also occur following deposition of foreign substances in the skin. Examples are tattoos where black carbon is introduced into the dermis and melanosis following prolonged massive arsenic medication in which melanin pigment accumulates around the arsenic. Argynia is a similar phenomenon and follows ingestion or internal application of silver containing medications. Likewise, chrysiasis describes the pigmentation that follows the use of gold containing medications. Yellowish discoloration of the skin from carotene (aurantiasis) is observed following consumption of large amounts of carrots or oranges.

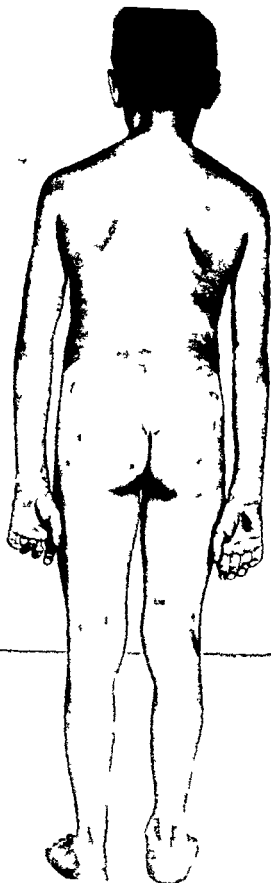


Fig. 139. Ligo
showing symmetrical
pigmented patches. Spontaneous
cure in the form of
ocular depigmentation
to be seen on those parts of
back which have been
exposed to sunlight.



XV. SKIN DISEASES OF THE LIPS AND MUCOUS MEMBRANE OF THE MOUTH

Abnormalities of the Mucous Membranes of Minor Clinical Significance

The so-called Fordyce disease actually is only an accumulation of yellowish, cobblestone like, ectopic sebaceous glands on the inner side of the lips and the mucous membranes of the cheeks. *Lingua geographica*, also called *exfoliatio areata linguae*, is characterized by roundish, slightly hyperkeratotic patches with central flattening of the papillae. Black tongue, or hairy tongue, shows hairlike, brownish hyperkeratoses of the papillae of the posterior part of the tongue, it is perhaps associated with smoking, nutritional imbalance and *Monilia* infection.

Stomatitis (Inflammation of the Mucous Membrane of the Mouth)

Plaut-Vincent Stomatitis (Trench Mouth)

This is an ulcerative stomatitis with purulent necrotic membranes and an acid odor. A combination of fusiform bacilli and coarse *Spirochaeta refringens* are seen in stained smears or under the dark field microscope.

Treatment: Penicillin effects a quick cure.

Monilial Stomatitis (Thrush)

There are whitish spots with a red margin on the mucous membranes, the membranes are formed by colonies of *Candida albicans*. Oral moniliasis occurs in debilitated and undernourished persons, in newborns and infants, and following protracted treatment with broad spectrum antibiotics, the so called "mycins".

Treatment: The underlying condition should be corrected. The mouth is rinsed with a mild antiseptic solution, or 1 per cent aqueous gentian violet solution is applied. Large amounts of vitamin B and C are added.

Stomatitis Due to Avitaminosis

Several classic avitaminoses are associated with stomatitis. Examples are the vitamin C deficiency, scurvy, the niacin deficiency, pellagra, and pernicious anemia (Hunter's glossitis) with disturbance of vitamin B₁₂ absorption. Large amounts of vitamin C plus vitamin B complex are a favorite treatment in all cases of stomatitis and are often effective, although outspoken cases of avitaminosis have become a rare occurrence.

Discoloration of the Gums Due to Bismuth, Lead and Mercury (Fig 141)

Parenterally administered bismuth and mercury are excreted on the mucous membrane as sulfides, especially in cases of poor oral and dental hygiene when hydrogen sulfite compounds are present in the mouth. Lead workers develop a lead line. In mild cases, the mucous membranes show a gray or blackish discoloration and in more severe cases a stomatitis develops around these deposits.

Treatment Good oral and dental hygiene and the use of mild antiseptic rinses for the mouth are recommended for patients receiving injections of bismuth or mercury.

Aphthous Stomatitis (Canker Sores)

Aphthae are painful oval erosions, pinhead to lentil sized, with a yellowish fibrinous membrane; they occur singly or in groups. Aphthous stomatitis may be acute and extensive and associated with fever, or it may be chronic and recurrent. The etiology is unknown but a virus is suspected. In some instances food allergy is a definite factor.

Treatment Elimination of provocative foods, if such are known, the use of Aureomycin lozenges, local application of silver nitrate solution, administration of vitamins and improvement of nutritional imbalance are recommended.

Allergic Stomatitis

Stomatitis and increased salivation may be caused by hypersensitivity of the mucous membranes to dentures, dental fillings and mouth washes.

Treatment consists of elimination of the causative agents.

Cheilitis (Inflammation of the Lips)

Hypersensitivity to lipstick leads to eczematous allergic inflammation of the lips. The photodynamic action of red dyes in lipsticks may also cause cheilitis. In cheilitis glandularis the deeper parts of the lips and their mucous glands are involved, its cause is unknown. In cheilitis granulomatosa there is a tuberculoid inflammatory granulation tissue, possibly tuberculous. Perleche (angular stomatitis) produces inflammatory fissures of the corners of the mouth caused by various factors such as vitamin B or iron deficiencies, with secondary streptococcal or monilial infection. Papules of secondary lues at the corners of the mouth may cause a similar clinical picture (see Fig 151).

Treatment Large amounts of vitamin B-complex plus vitamin C are given and iron is prescribed when indicated, for local treatment neomycin, bacitracin, Aureomycin, Terramycin or erythromycin ointments are recommended combined with hydrocortisone in more resistant cases.

Fig 141 *Bismuth lines of gums.* There is a gray discoloration of the gingiva around the teeth and a beginning bismuth stomatitis



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Cheilitis solaris occurs after intensive irradiation with sunlight, a diffuse inflammation of the lips with redness and scaling develops, especially when the lips have been dried out by wind and cold weather. It may be prevented by protecting the lips with sun protective lipstick or with a protective ointment such as yellow Vaseline or unguentum simplex.

Treatment Mild antiseptic ointments are used as recommended for dermatitis (see page 134).

Leukoplakia

In leukoplakia there are whitish patches on the lips and mucous membranes of the mouth and tongue, with slight inflammation and hyperkeratosis. After several years' duration, sometimes sooner, a leukoplakia may become verrucous and turn into a squamous cell carcinoma. The etiology of leukoplakia is not known, although smoking seems to favor its development. Lichen planus of the mucous membrane may turn into leukoplakia. Mucous plaques of secondary lues may resemble leukoplakia. Therefore, as in most oral lesions, an examination for syphilis is indicated. Since leukoplakia is a precancerous condition, the patient must be regularly checked so that malignant degeneration can be recognized early.

Treatment The smooth, whitish leukoplakia requires no treatment, except when the patient so desires. Vitamin A in large amounts has been helpful, perhaps best in the form of troches (Vi-Dom-A tablets, 150,000 units dissolved slowly in the mouth). Verrucous leukoplakia should be treated either by electrocoagulation (with or without radium), or surgically.

Alterations of the Mucous Membrane of the Mouth in Skin Diseases

The exanthems of infectious diseases cause mucous membrane changes as exemplified by Koplik's spots in measles or a dark red tongue in scarlet fever (raspberry tongue). Chickenpox and smallpox involve the mucous membranes in the form of smaller or larger erosions. Syphilis may involve the mucous membranes as a primary chancre with swelling of the regional lymph nodes or as mucous plaques in the secondary stage (see Figs 144 and 152). Ulcerated gummas occasionally are located on the hard palate or pharynx, they heal with scar formation. Syphilitic papules on the corners of the mouth, resembling perleche (Fig 151) and leukoplakia may again be mentioned. Tuberculosis may present a soft ulcerated primary complex on the gingiva or on the tonsils and lupus vulgaris may be located on the mucous membranes of the mouth or nose. Herpes simplex frequently occurs on the lips (Fig 151) and sometimes on the oral mucosa. Herpes zoster involving the maxillary branch of the trigeminal nerve, erythema multiforme, and also dermatitis herpetiformis may involve the

lips and the mouth Pemphigus vulgaris not infrequently originates as an extensive erosive stomatitis, which may accompany all subsequent eruptions In lupus erythematosus, whitish, hyperkeratotic and slightly inflamed lesions are found on the mucous membranes whereas netlike, cobblestone like, whitish plaques or streaks are the very resistant oral manifestation of lichen planus (Fig 68) Hemangiomas and lymphangiomas often occur on the lips and the adjacent buccal mucous membranes

XVI VENEREAL DISEASES

Venereal diseases are a diverse group of contagious diseases which are similar in only one respect they are primarily spread by sexual intercourse. In gonorrhea this route of contagion results from the predilection of the gonococcus for the mucosa of the urogenital tract. Syphilis requires close person to person contact for viable transfer of the treponemas. In civilized countries infection occurs chiefly during the intimacies of love. Among primitive countries syphilis may exist as an endemic disease with widespread extragenital transmission.

Fig 142 *Treponema pallidum*



Fig. 143 *Syphilis chancre of the penis* Round necrotic ulcers on the inside of the foreskin



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Fig 142 *Treponema pallidum*



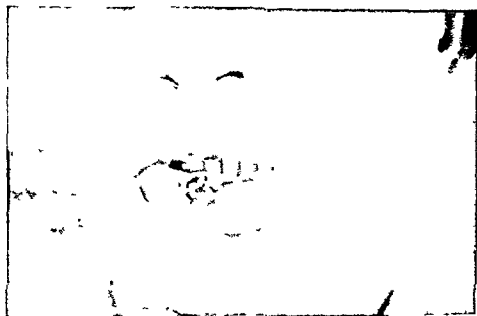


Fig 144 *Syphilitic chancre of the lower lip*

Fig 145 *Syphilitic roseola* →

The incidence of the venereal diseases shows marked fluctuations, being greatest during times of war and revolution as a result of the social disorganization that always accompanies such tragedies. After both World Wars I and II great increases in syphilis occurred in the war ravaged countries. However, syphilis now has come under control and has become a disease infrequently seen today. In the United States it has become so rare that most present day medical students never have the opportunity to observe a case of early syphilis. This dramatic disappearance of syphilis is probably related to the efficacy and widespread use of penicillin. Gonorrhea, however, is still prevalent in all civilized countries and is by far the most common venereal disease. Chancroid is not uncommon in the southern United States, but lymphogranuloma venereum and granuloma inguinale are rare in the United States.

In all venereal diseases the physician has the opportunity and responsibility to practice preventive medicine. Sexual contacts and particularly marital partners require an examination and treatment as indicated. The public health authorities must be informed of all new cases as required by law.

One further word of caution. Less experienced physicians tend to assume that every sore or lesion of the genitalia is venereal. In a civilized society the opposite is usually true. The skin of the genitalia is not immune to herpes simplex, folliculitis, and a host of other dermatologic conditions. Misdiagnosing such conditions as venereal will produce much unnecessary fear and anguish.

Syphilis

Primary Syphilis (Figs 142 to 144)

The cause of syphilis is the *Treponema (Spirochaeta) pallidum*, discovered in 1905 by Schaudinn and Hoffmann. The spirochetes gain entry into the body via tiny abrasions of the skin or mucosa. The chancre, the primary lesion of syphilis, usually appears after an incubation period of 3 to 4 weeks. In exceptional cases the chancre may appear as early as 10 days or as late as 5 weeks after intercourse. At first an indurated papule, the lesion promptly erodes to form the characteristic chancre, an indurated ulcer. Palpation of such lesions with a gloved finger produces the sensation of a coin sized nodule of hard rubber consistency lying within the skin. Chancres may be solitary or multiple, varying in diameter from a few millimeters to several centimeters. The larger lesions leave obvious scars. Although chancres are most common on the genitalia they are occasionally found about the anus (in homosexuals), the lips, the nipple or the fingers. Failure to detect a primary lesion occurs in about one half of the cases in women but in only a small proportion of male cases.

Regional lymphadenitis develops 2 or 3 weeks after the appearance of the chancre. The enlarged glands are painless, the glands remain discrete and do not

become adherent to the overlying skin. Chancres of the lips produce marked swelling of the submandibular nodes. Six weeks after the infection is contracted antibodies appear in the blood and the various serologic reactions become positive. The first reaction to become positive is the complement fixation test, next, the flocculation reactions, and finally, Nelson's specific treponema immobilization test. The clinical diagnosis must be confirmed by demonstration of spirochetes or by serologic reactions. In the first few weeks after the appearance of the chancre the serologic tests are negative and the diagnosis can be established only by demonstrating the spirochetes with a dark field microscope. To postpone treatment until the serologic tests become positive is dangerous, as this invites further transmission of the disease. The best way to demonstrate the spirochetes is by immediate dark field examination of the exudate squeezed out from the ulcer. This procedure must be carried out by a physician especially trained in the technique. Local antiseptics and penicillin or other antispirechetal agents interfere with the demonstration of spirochetes or make it altogether impossible. Compresses with physiologic saline are ideal for preparing a lesion for dark field examination. Interpretation of a positive serologic test for syphilis is at times a difficult matter because a large variety of diseases and conditions may produce transitory or prolonged positive reaction. Such a positive serologic test is known as a biologically false positive reaction and may result from infectious mononucleosis, vaccination, malaria and numerous other diseases.

Secondary Stage of Syphilis (Figs 145 to 152)

Even without treatment chancres heal in a few weeks. At this time or shortly afterwards the roseola, the first sign of secondary syphilis, makes its appearance. This exanthem consists of numerous small bright red spots scattered over the trunk, extremities, neck and forehead. Papular lesions may appear during the course of the eruption and these at times form fine scales. Itching is absent. Prior to or during the early stages of the exanthem mild systemic symptoms are a frequent but not invariable occurrence. Most commonly these consist of malaise, headache, a low grade fever and generalized lymphadenopathy. Systemic symptoms may be absent and the roseola may be overlooked by the patient. After the early roseola has disappeared a recurrent roseola may follow with grouped papules that are more pronounced. Within a period of several weeks or months further variable skin manifestations develop. On the palms and soles there may be macular or partially papular lesions with slight or massive psoriasis like scaling. About the genitalia and anus, as well as in other intertriginous areas, the lesions are in the form of large papules, the surfaces of which are generally eroded, these are known as condylomata lata. The moist

Fig 148 *Secondary syphilis of the soles* with brownish red papules

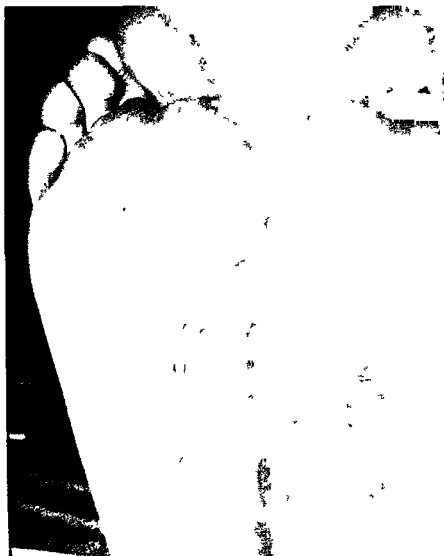




Fig. 149 *Populosisquamous*
syph. of the soles

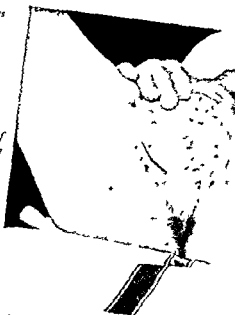


Fig. 150 *Popular syphilid of*
anx and genita's (condylomata
lata)

Fig. 151 *Syphilitic papules*
of corner of mouth and nostrils

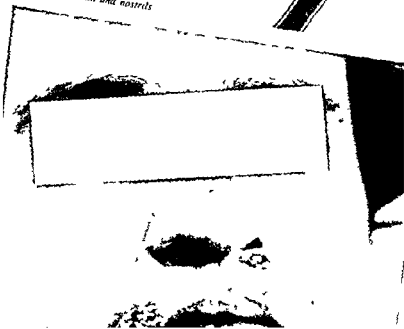




Fig 153 *Seipogenous tertiary syphilis* In the periphery there are crusted nodules
in the center atrophy and scars



Fig. 153 *Serpiginous tertiary syphilis*. In the periphery there are crusted nodules in the center atrophy and scars



Fig. 155 Mesoaortic atherosclerosis x ray shows widening of the aortic arch

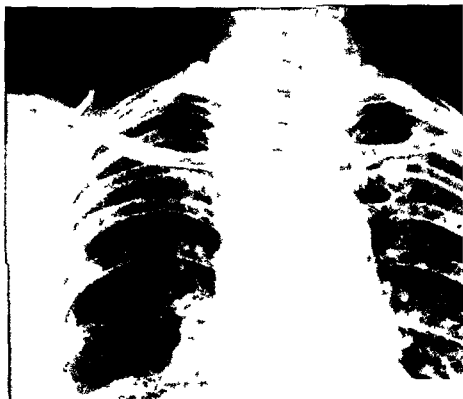


Fig 156 *Malum perforans in tabes* The fistula penetrates
to the bone and is surrounded by hyperkeratosis



surface of these condylomas teems with spirochetes and they are consequently highly infectious lesions. These syphilitic plaques must be distinguished from the condylomata acuminata or venereal warts which are harmless and are caused by a virus (see page 81). Syphilitic erosions tend to appear at the corners of the mouth and in the nasolabial folds. In contrast to banal fissures syphilis produces eroded papules. The buccal mucosa, gums, tongue and larynx frequently show reddish papules. A patchy alopecia sometimes occurs in the

- -

and years later suggest a previous episode of syphilis.

Systemically some degree of meningitis with severe headaches may be apparent. At times periostitis may cause considerable pain in the tibia or other long bones.

Diagnosis is substantiated by serologic tests which are always positive in the secondary stage of syphilis. The spirochetes can frequently be demonstrated in exudates from secondary lesions.

Third State of Syphilis (Tertiary Lues) (Figs 153 and 154)

The signs and symptoms of tertiary syphilis may appear at any time from several months to many years after the secondary stage. Almost any tissue or organ may become involved. In the skin the lesions are grouped and localized since immunologic changes restrict the action of the spirochetes. The papular serpiginous syphilid consists of large reddish brown papules arranged in groups or in irregular lines. In the ulceronodular serpiginous syphilid the papules are more massive and undergo ulceration with central scarring. Further peripheral extension and central healing produces lesions with a variety of different shapes with round or polycyclic borders and kidney shaped ulcerations. Scrofuloderma like lesions may occur. Gummas of the skin are walnut sized subcutaneous infiltrates which often ulcerate after a variable period of time. The end result is a deep ulcer with punched out margins. Gummas of the palate produce perforating ulcers leaving an almost diagnostic defect.

Systemically there may be syphilitic arthritis, chorioretinitis and gummas of bone as well as involvement of the liver, kidneys or lungs. Highly significant is the involvement of blood vessels by syphilis. This is a late manifestation usually becoming clinically evident only two or three decades after the disease is contracted. The most common lesion is mesaortitis (Fig 155) with replacement of the elastic fibers by scar tissue. The aorta dilates and the aneurysm so formed may cause aortic insufficiency or occlusion of the coronary arteries. Rupture of the aneurysm or sudden occlusion of the coronary vessels produces sudden

death According to Bruusgaard, 17 per cent of untreated syphilitics develop mesaortitis

The serologic tests in tertiary syphilis of the skin are usually positive, but occasionally they are negative in tertiary syphilis of internal organs The dark field examination of tertiary lesions is fruitless Occasionally it is possible to demonstrate spirochetes in especially stained tissue sections of tertiary lesions

Neurosyphilis

Five to 10 years after the onset of syphilis its effect on the parenchyma of the central nervous system may become evident These effects are usually classified in two syndromes, tabes dorsalis and general paresis In addition, gummas within the central nervous system may mimic a brain tumor Syphilitic vascular disease may resemble an endarteritis or arteriosclerosis and at times produce acute hemi- or paraplegias as well as chronic brain degeneration The history serologic tests and cerebrospinal fluid changes must all be evaluated in making the diagnosis

General Paresis

Personality changes are the earliest manifestation of general paresis Patients become careless, unreliable, forgetful and euphoric The picture may be quite variable and at times there may be megalomania, depression and episodes of unconsciousness Ultimately, progressive dementia appears Reflex and pupillary changes often occur, usually the pupils react poorly to light Examination of the spinal fluid is necessary, it reveals increases in the number of cells and total protein as well as an abnormal colloidal gold curve The serologic tests of the spinal fluid are positive

Tabes Dorsalis

Tabes dorsalis results from degeneration of the posterior spinal nerve roots and posterior columns of the spinal cord Signs and symptoms develop and progress gradually over many years The tendon reflexes of the lower extremities disappear A loss of position sense occurs and the typical ataxic gait results The Romberg sign inability to stand unsupported with eyes closed and feet together is positive The finger-to nose test presents difficulties The pupils frequently react poorly or not at all to light while still reacting to accommodation Unilateral or bilateral optic atrophy is often associated with tabes dorsalis Severe, sharp lightening pains may occur, most frequently in the lower extremities Visceral crises of abdominal distress and pain usually with vomiting can mimic almost any abdominal disorder Trophic changes are felt to result from peripheral nerve damage and are exemplified by Charcot joints and per-

forating ulcers of the foot (malum perforans) (Fig 156) Ultimately cachexia sets in. Serologic examination of blood and spinal fluid confirms the diagnosis, although occasionally the serologic reactions are negative in tabes.

Latent Syphilis

Syphilis produces symptoms only intermittently. For long periods it smolders unnoticed and then is referred to as latent syphilis. It is customary to describe latency in the secondary stage as early latent, and in the tertiary stage it is called late latent lues. When a physician detects a positive serologic test for syphilis in the course of a routine examination the patient should be questioned as to previous venereal disease, serologic tests and current symptoms. Examination of the skin, genitalia, reflexes and mental status may reveal evidence of old or fresh syphilis. Chest x ray for mesaortitis, as well as a spinal fluid examination to rule out neurosyphilis are indicated. In the absence of a history or physical findings of syphilis it is mandatory to have the serologic tests repeated at least once. If the results are doubtful it is advisable to have them checked by several reliable laboratories. Nelson's Treponema immobilization test eliminates non-specific serologic reactions; however, this test is expensive and is performed in only a small number of laboratories.

Treatment of Syphilis

Penicillin is the drug of choice, arsphenamine, bismuth and mercury are mainly of historic interest now.

Various treatment routines have been recommended. For primary syphilis 15 daily injections of 600,000 units of procaine penicillin are adequate, or 4,800,000 units of penicillin with aluminum monosterate in oil (PAM) given in doses of 1,200,000 units at intervals of 2 to 7 days. For unreliable patients, or for mass treatment a single injection of 2,400,000 units of delayed acting penicillin (benzathine penicillin G) are recommended. Tertiary lues and neurosyphilis may require somewhat more prolonged treatment giving a total of 15,000,000 units of penicillin. Fever therapy is sometimes combined with penicillin in the therapy of neurosyphilis.

If penicillin treatment is contraindicated because of allergic sensitivity to the drug other antibiotics should be given, e.g. tetracycline, Aureomycin or Terramycin. Early and latent syphilis are treated orally with at least 3 to 4 g daily for 10 to 12 days and neurosyphilis or cardiovascular syphilis for at least 15 days.

The so-called Herxheimer reaction, consisting of fever, chills, malaise, headache, myalgia, arthralgia, and a transient exacerbation of the skin lesions (exanthem and fever) is a common reaction to the treatment of syphilis. It is harmless in primary and secondary syphilis but the patient should be advised

that it may occur. In cardiovascular lues and neurosyphilis the Herxheimer reaction was feared especially in former times when arsphenamines were the choice of drugs. American authors believe that there is only a small hazard that such reactions will follow treatment with penicillin; they consider preparatory treatment with bismuth or the use of cautious initial doses of penicillin in general unnecessary. European syphilologists, however, are concerned with untoward effects of the Herxheimer reaction and the so called therapeutic paradox (aggravation of symptoms) especially in cardiovascular, central nervous system and tertiary lues. In order to minimize such responses from penicillin, rare as they may be, they recommend preparatory treatment with bismuth. A satisfactory course of bismuth therapy consists of a series of 15 to 20 injections of 1.5 cc. bismuth subsalicylate (Stabisol) given at semiweekly intervals. As a first dose only 0.5 cc. is given followed by 1.0 cc. for the next injection. Complications of bismuth therapy are nephrosis, bismuth line of the gingiva and stomatitis (Fig. 141). In tertiary lues preparatory treatment with potassium iodide followed by bismuth is recommended where rapid regression of the gummatous lesions may lead to damage of vital organs or to cosmetic defects. Serologic reactions in primary and secondary lues often become negative only several months after treatment. The blood should be checked at monthly intervals until it becomes negative and thereafter every third month. After the second year blood checks every 6 months are recommended and between 5 and 10 years after the infection once a year. While reversal of the serologic reactions to normal is a reassuring sign of effective treatment, it does not always happen. In spite of adequate treatment and apparent clinical cure the serologic tests, especially in the latent and tertiary stages and in neurosyphilis, may remain positive. If the patient has had adequate treatment it is advisable not to try for a reversal of the positive serologic reaction by repeated courses of treatment.

The best time to examine the spinal fluid is 2 years after infection, because at that time changes accompanying secondary lues have disappeared and positive reactions are considered a sign of impending neurosyphilis. At subsequent examinations a neurologic examination and fluoroscopy of heart and aorta are indicated.

Congenital Syphilis

Spirochetes from an infected mother can cross the placenta to attack the fetus. The resulting disease may cause abortion, birth of an obviously syphilitic infant, or an infection which is first manifest after some years (late congenital syphilis). If the mother has active early syphilis, abortion is the usual course. If the mother's syphilis is in the later stages a living but sick child is the rule. The

infant may be fortunate enough completely to escape infection if the mother has late latent lues

There is no primary lesion in congenital lues since the dissemination occurs *via* the blood stream At or shortly after birth various exanthems appear corresponding to the secondary stage of syphilis Vesicles and bullae of the palms and soles are a frequent finding Rhinorrhea and hoarseness are a consequence of mucous membrane involvement Bony changes are an important early sign Osteochondritis of the long bones may lead to epiphyseal separation Rhagades about the lips paronychia and diffuse alopecia are common

Children with severe active congenital syphilis are debilitated The generalized spread of spirochetes of which splenomegaly and hepatomegaly are signs leads to the high mortality rate of 30 per cent, even with proper treatment Late congenital syphilis may not be detected until 5 to 20 years after birth, at this stage the classic Hutchinsonian triad of Hutchinson's teeth (Fig 157), eighth nerve deafness and interstitial keratitis is sometimes encountered The dental malformations involve mainly the central upper incisors Usually these show an almond shaped notching but they may be peg or barrel shaped All these changes are caused by defective development of the dental foci during pregnancy

Fig 157 *Hutchinson's teeth in congenital lues* with semilunar indentation of the upper central incisors



that it may occur. In cardiovascular lues and neurosyphilis the Herxheimer reaction was feared, especially in former times when arsphenamines were the choice of drugs. American authors believe that there is only a small hazard that such reactions will follow treatment with penicillin, they consider preparatory treatment with bismuth or the use of cautious initial doses of penicillin in general unnecessary. European syphilologists, however, are concerned with untoward effects of the Herxheimer reaction and the so called therapeutic paradox (aggravation of symptoms) especially in cardiovascular, central nervous system and tertiary lues. In order to minimize such responses from penicillin, rare as they may be, they recommend preparatory treatment with bismuth. A satisfactory course of bismuth therapy consists of a series of 15 to 20 injections of 1.5 cc bismuth subsalicylate (Stabisol) given at semiweekly intervals. As a first dose only 0.5 cc is given, followed by 1.0 cc for the next injection. Complications of bismuth therapy are nephrosis, bismuth line of the gingiva, and stomatitis (Fig 141). In tertiary lues preparatory treatment with potassium iodide followed by bismuth is recommended where rapid regression of the gummatous lesions may lead to damage of vital organs or to cosmetic defects. Serologic reactions in primary and secondary lues often become negative only several months after treatment. The blood should be checked at monthly intervals until it becomes negative and thereafter every third month. After the second year blood checks every 6 months are recommended and between 5 and 10 years after the infection once a year. While reversal of the serologic reactions to normal is a reassuring sign of effective treatment, it does not always happen. In spite of adequate treatment and apparent clinical cure the serologic tests especially in the latent and tertiary stages and in neurosyphilis may remain positive. If the patient has had adequate treatment it is advisable not to try for a reversal of the positive serologic reaction by repeated courses of treatment.

The best time to examine the spinal fluid is 2 years after infection, because at that time changes accompanying secondary lues have disappeared and positive reactions are considered a sign of impending neurosyphilis. At subsequent examinations a neurologic examination and fluoroscopy of heart and aorta are indicated.

Congenital Syphilis

Spirochetes from an infected mother can cross the placenta to attack the fetus. The resulting disease may cause abortion, birth of an obviously syphilitic infant, or an infection which is first manifest after some years (late congenital syphilis). If the mother has active early syphilis, abortion is the usual course. If the mother's syphilis is in the later stages a living but sick child is the rule. The



Fig 158 *Gonococci* Intracellular Gram negative diplococci in leukocytes

both extra and intracellularly (Fig 158) and can be found in the urethral and cervical discharge. When cultured the gonococcus forms small grayish colonies on enriched media.

Gonorrhea in the Male

Uncomplicated Gonorrhea

After an average of 3 to 5 days following contagious sexual intercourse burning on urination is noted and at the same time a urethral discharge appears. At first serous it soon becomes purulent and the leukocytes usually teem with gonococci. This is the stage of anterior urethritis involving only the portion of the urethra in front of the sphincter. If the urine is collected in two containers (two-glass test) only the first portion is cloudy.

If untreated after 2 to 3 weeks the disease involves the posterior urethra. Sphincter spasms may then occur. In posterior urethritis the discharge runs back into the bladder and in the two-glass test both urine specimens will be cloudy. Infection of the posterior urethra sets the stage for the acute complications of gonorrhea: prostatitis, seminal vesiculitis and epididymitis. Even in the absence of treatment the acute symptoms gradually abate over a period of several weeks. A slight mucopurulent discharge is the only outward sign of chronic gonorrheal urethritis. This may last several months during which time the patient remains infectious and liable to develop complications.

Any manifestation of tertiary lues such as serpiginous exanthems, gummata of skin, bones or internal organs, cardiovascular syphilis, tabes and paresis may occur in congenital syphilis. Neurologic signs in congenital lues usually appear between the ages of 15 and 20. The serologic tests are positive in all cases of congenital lues and may remain positive for many years after successful treatment.

Treatment of Congenital Syphilis

In congenital syphilis too, penicillin is the drug of choice. Adequate treatment of the pregnant mother insures against congenital syphilis in the child. The syphilitic newborn can be cured by relatively small doses of penicillin, e.g., 200,000 units of procaine penicillin daily for 10 days. To be on the safe side it may be well to repeat this course. In older children, a regimen similar to that for adults is used, but the daily doses are somewhat smaller. The excellent results and small danger of penicillin therapy suggest that a child of a syphilitic mother should receive a course of penicillin even in the absence of luetic symptoms. This will insure against the development of late congenital lues.

Other Related Treponemal Diseases

Yaws, pinta and bejel are caused by treponemata which are morphologically closely related or identical to *Treponema pallidum* of syphilis. These diseases also show the same serologic reactions as syphilis. Clinically these disorders are distinctive and for this reason it is believed that they are caused by spirochetes different from, but closely related, to the *Treponema* causing syphilis.

Yaws (frambesia) is an endemic tropical disease, caused by *Treponema pertenue* and characterized by papular and markedly hypertrophic as well as ulcerated lesions in the primary and secondary stages. In the tertiary stage gummatous and other destructive and mutilating lesions appear, juxta-articular nodes are relatively frequent.

Bejel is a form of treponematoses endemic among certain Arabs and is either closely related or identical to syphilis.

Pinta is a treponemal disorder found only in certain South American countries. It produces bizarre pigmentary changes in the skin which are often striking. Atrophic and destructive late skin lesions can occur, as may neurologic and cardiovascular involvement.

Treatment Yaws, bejel and pinta respond well to penicillin.

Gonorrhea

The cause of gonorrhea is the gonococcus discovered by Neisser in 1879. It is a Gram negative Diplococcus which tends to be arranged in small clusters,



Fig. 158 *Gonococci* Intracellular Gram negative diplococci in leukocytes

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Genital Complications of Gonorrhea in the Male

Today, complications in men are infrequent. Epididymitis or prostatitis occur in only about 3 per cent of treated cases, whereas before the development of effective antibacterial therapy complications assumed a frequency of 30 per cent.

Gonorrheal prostatitis Involvement of the prostate results in indefinite, cramplike pains noted in the lower abdomen and perianal area, as well as sphincter cramps and backache. In severe cases there is urinary retention and fever may occur. Rectal examination reveals an enlarged and tender prostate. The prostatic secretion, expressed by massage, contains numerous leukocytes and gonococci. However, prostatic massage should not be done in the acute case, even for diagnostic purposes.

Gonorrheal epididymitis Invasion of the vas deferens and epididymis by gonococci produces an acute painful swelling of the spermatic cord and epididymis. It is difficult to distinguish the tensely swollen epididymis from the softer, less swollen testicle which is generally edematous because of the nearby inflammation. After several days the swelling decreases and the firm epididymis may be easily palpated. Fever is usually present. Gonorrheal epididymitis, unlike tuberculous epididymitis, does not result in abscesses, fistulas, or adherence to the overlying skin; bilateral epididymitis may result in a permanent azoospermia.

Seminal vesiculitis Inflammation of the seminal vesicles causes symptoms similar to those of prostatitis. Rectal examination reveals swelling and tenderness of the region just above the prostate where they are located.

Other Complications

There are several other, relatively rare, complications of gonorrhea in the male. Lymphadenitis of the inguinal nodes, sometimes associated with lymphangitis of the dorsal surface of the penis, can occur. There may also be infection of Cowper's and Littre's glands as well as of the paraurethral sinuses in the fossa navicularis. Urethral stricture is currently felt to be chiefly the result of the formerly used local treatment with injurious chemicals, rather than of the infection itself. Gonorrheal infection of the male rectum may result from homosexual practices.

Gonorrhea in the Female

Uncomplicated Gonorrhea

In women the urethra and cervical canal are primarily effected, but the discharge may infect the rectal mucosa as well. Symptoms are mild, in the early stages there is burning on urination and a profuse, purulent vaginal discharge.

Rectal gonorrhea often produces no symptoms but may manifest itself by anal pruritus and by spastic discomfort of the rectum. However, in many cases of uncomplicated female gonorrhea the symptoms are so mild that the disease is detected only when the sexual partner becomes infected. The gonococci may be demonstrated in urethral as well as cervical smears. Urethral smears should be performed prior to urination or catheterization. Smears from the rectum, best obtained with a proctoscope, may also demonstrate the organism. In order to detect chronic gonorrhea in the female it is often necessary to make smears on several occasions. The increased secretions in the cervical canal after menstruation heighten the chances of obtaining a positive smear in chronic gonorrhea. For this reason suspicious cases should have smears performed immediately after the menstrual period.

Genital Complications of Gonorrhea in the Female

Complications have become uncommon. Endometritis and adnexitis occurred in only 2 per cent of cases seen in recent years at the clinics in Zurich, Switzerland.

Gonorrheal adnexitis and endometritis Infection of the uterine cavity and Fallopian tubes causes acute symptoms with severe abdominal cramps and pain which may mimic an acute appendicitis. Bimanual pelvic examination reveals

Endometritis causes an increased purulent discharge from the cervix and often metrorrhagia. Bartholinitis is another complication and may cause redness of the introitus and swelling of Bartholin's glands in one or both labia majora.

Gonorrheal vulvovaginitis Gonorrheal infection of a prepubertal girl (through rape or through contagion from an infected person sharing the same bed) produces a vulvovaginitis because at this age the mucosa of the vagina and vulva lacks resistance to the gonococcus.

Extragenital Complications of Gonorrhea

Gonorrheal arthritis Hematogenous dissemination of gonococci can cause joint infection. This septic complication may appear as a relatively benign serous polyarthritis or as a purulent process which usually affects only one joint and often leads to its destruction. In recent years arthritis complicated only 0.4 per cent of cases of gonorrhea seen in Zurich, Switzerland. However, a rheumatoid like arthritis sometimes supervenes in otherwise uncomplicated cases of gonorrhea, cured by penicillin.

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Gonorrheal conjunctivitis In infants and old people gonorrhea may infect the eye to produce a purulent conjunctivitis. In young adults where gonorrhea is most common, gonorrheal conjunctivitis is rarely seen. Newborn infants acquire their infection from the mother's birth canal. In order to prevent gonorrheal conjunctivitis (*ophthalmia neonatorum*) an antiseptic is instilled into the eyes of the newborn. Customarily, 1 per cent silver nitrate is used, but solutions of penicillin or other antibiotics have also been recommended.

Other Complications

Infections of the bladder or kidney are rare, although the bladder mucosa frequently comes into contact with pus containing gonococci.

Treatment of Gonorrhea

As a rule gonococci are very sensitive to penicillin. Two or preferably three daily injections of 600,000 units of procaine penicillin will cure most cases of gonorrhea, in the female as well as the male. In cases of gonorrheal epididymitis, prostatitis, arthritis, adnexitis, conjunctivitis, and vulvovaginitis of young girls daily injections of penicillin should be continued until the symptoms subside. *Rarely are more than four to five injections needed.*

In epididymitis, acute prostatitis, adnexitis and arthritis bed rest is indicated. Penicillin, together with proper supportive care, usually cures these complications in a matter of days. Topical therapy has been completely replaced by specific antibacterial treatment. For a number of years the sulfonamides were popular, but the development of sulfa resistant gonococci ended the usefulness of these drugs. At present penicillin is the antibiotic of choice. Textbooks generally state that penicillin resistance among gonococci does not occur, but this no longer holds true. Where penicillin resistance has been observed and in cases of penicillin allergy, one of the tetracyclines or chloramphenicol may be used. 1 to 2 g daily in divided doses for 5 days is generally curative.

Gonorrhea is a highly infectious disease and a patient's marital partner should be treated at the same time. Simultaneous treatment of sexual partners is highly advisable even if the infection cannot be demonstrated. Otherwise gonorrhea may be passed back and forth in a ping pong manner.

Follow up care in men should include smears taken at two or three return visits several days apart. In women several smears should be taken preferably right after the menstrual period. Because penicillin administered for gonorrhea may mask a simultaneously acquired syphilis patients with gonorrhea are advised to have a serologic test for syphilis 6 to 8 weeks later.

Chancroid (Soft Chancre)

Chancroid has become a rare disease in Europe and the northern parts of the United States. The genital ulcers of chancroid appear 3 to 14 days after contact and may be single or multiple. They are tender and have a dirty necrotic base and usually show less induration than the syphilitic chancre. The regional lymph nodes, usually the inguinal nodes, often are swollen; the inflammation may spread to the overlying skin and perforation of the lymph nodes may occur.

Smears from the ulcer base will usually demonstrate clusters of the Gram negative rods of *Hemophilus ducreyi*, the causative organism. Bacterial culture as well as various skin tests are available to confirm the diagnosis. Inoculation of the pus at another site generally leads to formation of a new lesion.

Treatment. Sulfadiazine and streptomycin are the drugs of choice. 10 g of sulfadiazine is given four times daily for 7 days, or daily injections of 1 g of streptomycin are given for 1 week. Chloromycetin, Aureomycin, Terramycin and tetracycline, 250 mg four times daily for 1 week, are also effective but may mask an accompanying syphilis and are best reserved for exceptional cases. Local use of mildly antiseptic soaks, such as dilute potassium permanganate, is of benefit.

Lymphogranuloma Venereum

This fourth venereal disease is most common in tropical areas, although it is not unknown in the United States. After an incubation period of 2 to 6 weeks a rather trivial primary lesion appears in the form of a small herpes-like ulcer on the prepuce or labia. Frequently the primary lesion escapes notice. Thereafter there is progressive swelling and suppuration of lymph nodes to form large buboes which may break down to form multiple draining fistulas. The destruction of lymph nodes and consequent scarring may lead to chronic swelling or elephantiasis of the genitalia (esthiomene), as well as rectal strictures and anal fistulas. According to Sonck, a frequent minor complication is an erythema multiforme-like eruption of those parts of the skin which are exposed to the sun.

The causative organism is a filtrable virus which can be transmitted to mice, monkeys and chicken embryos. Diagnosis is best confirmed by the *Frei test*. This consists of the intradermal inoculation of an antigen, formerly sterilized pus but now prepared from yolk sac cultures of the virus. A tuberculin-type response with a reddened papule over 5 mm in diameter, obtained after 48 to 72 hours, represents a positive test.

Treatment. Aureomycin or other broad spectrum antibiotics, 1 g daily for 10 or more days, give good results in early cases. Sulfadiazine may also be used. In late cases with elephantiasis and rectal strictures the results are far from satisfactory.

Granuloma Inguinale

This venereal disease also occurs chiefly in tropical and subtropical countries and in the southern parts of the United States. The primary lesion is a papule or vesicle which soon becomes ulcerated. Steady peripheral extension may lead to large ulcers of the penis and inguinal region, with piled up granulation tissue at the margins. The cause of granuloma inguinale has been ascribed to Donovan bodies and these can be demonstrated in the cells of Wright stained smears made from ulcer scrapings. Treatment with broad spectrum antibiotics is usually necessary to effect a cure, sulfonamides and streptomycin may also be used.

XVII. OTHER DISEASES OF THE GENITALIA

Nongonorrheal Urethritis in the Male (Urethritis Simplex)

A burning sensation upon urination and a purulent discharge somewhat less pronounced than in gonorrhea are signs and symptoms of nongonorrheic urethritis in the male. Often there is only a slight watery discharge, especially in the morning before urination. In the two-glass test the first urine specimen is slightly cloudy, and if the posterior portion of the urethra is involved the second specimen also. Smears show a few leukocytes, epithelial cells and mucus, and usually, but not always, a mixed bacterial flora consisting of Gram negative rods and Gram positive cocci. Prostatitis and epididymitis occur occasionally, but their course is less acute than in gonorrhea.

This form of urethritis may be due to various causes. Not infrequently a non-specific urethritis persists after gonorrhea. Occasionally Trichomonas is found in the male urethra, this is a saprophyte of the female usually found in the vagina but it may cause urethritis in the male. Rickettsia, the causative organism of pleuröpneumonia in cattle, has been found in 17 per cent of non-specific urethritis, this organism can be demonstrated only in cultures. Condiments, cold beverages, alcohol and sexual intercourse exert an unfavorable effect on the disease.

Treatment Spontaneous cure often occurs following abstinence from sexual intercourse and alcohol. In resistant cases tetracyclines are given, 250 mg four times daily for 4 days, or penicillin or sulfonamides may be administered. Instillation of 0.5 per cent silver nitrate or 5 per cent Argyrol into the urethra may be required. In resistant cases the possibility of a urethral stricture or other urethral lesions should be considered.

A rare syndrome is Reiter's disease, consisting of a nonspecific urethritis, conjunctivitis and arthritis, cutaneous lesions resembling those of keratosis blennorrhagica have been observed. It may be complicated or preceded by bacillary dysentery.

Treatment The condition usually is quite resistant to treatment but some cases respond well to adrenocorticotropin (ACTH) and cortisone.

Balanitis

Balanitis is an inflammation of the glans penis and the mucosal surface of the prepuce and is often accompanied by purulent discharge. The glans penis presents superficial erosions with polycyclic borders (balanitis erosiva) and the

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XVIII. TECHNIQUES OF SOME TESTS USED IN DERMATOLOGY

Patch Tests (Jadassohn and Bloch)

The material to be tested is applied in the proper dilution to a piece of gauze about 1 cm in diameter, the latter is placed on normal skin, covered with a slightly larger piece of cellophane and fixed to the skin with adhesive tape. Prepared patch test tapes are available, for example, Elastopatch and Dalmas patches. In case of hypersensitivity to adhesive tape, one may try Scotch tape or gauze soaked with collodion, or fixation of the patch test on the arm by means of a gelatin bandage as used for Unna's paste boot. The test material is removed after 24 hours and is read immediately, and again 24 and 48 hours later. Positive tests are characterized by an eczematous delayed reaction of papules or vesicles.

When a photodynamic or photoallergic action is suspected, a photopatch test is carried out. The substance to be tested, e.g., a plant or medication, is applied to the skin for several hours and the site so prepared is irradiated with direct sunlight for 1 to 2 hours or with an ultraviolet lamp. In the latter case it is advisable to remove the short ultraviolet rays with an appropriate filter or with ordinary window glass, which eliminates irradiation below 3200 Å. A similarly prepared site not exposed to light, serves as control. If the inflammation occurs only on the irradiated site, this indicates a photodynamic or photoallergic effect.

Scratch or Intradermal Test

In cases of cutaneous vascular allergy such as urticaria, infantile eczema, atopic dermatitis, asthma or allergic rhinitis, the substance to be tested must be brought in contact with the vascular apparatus of the cutis. Scratch testing is the most simple procedure, with an appropriate scratcher or a sharp knife short scratches about 5 mm long are produced on the inside of the arms, the environmental or food allergens or drugs are then applied. A positive result is manifested after 5 to 20 minutes by a wheal with pseudopods, surrounded by a red halo. Controls are always required, because a simple scratch may cause a wheal in urticaria factitia (dermographism). Intradermal tests are more sensitive. About 0.02 to 0.03 cc of a sterile solution of the allergen is administered by intradermal injection. Allergenic extracts can be bought commercially (Hollister-Stier, Benedict, etc.).

pseudo
reaction
control

biosis of fusiform rods and spirochetes. Monilia (*Candida albicans*) also may be the cause of balanitis. Diabetes mellitus is a contributing factor.

Treatment Soakings with potassium permanganate in a 1:10,000 solution are recommended, and application of powders containing Vioform or 5 per cent tannic acid, alternated with ointments containing 5 per cent Protargol or 1 per cent Rivanol in yellow petrolatum are useful. Underlying conditions such as diabetes must be corrected and personal hygiene should be stressed. Circumcision is advisable in recurrent cases.

Phimosis

Phimosis is a narrowing of the opening of the foreskin and may be congenital or may develop as an inflammatory condition following repeated attacks of balanitis. It interferes with urination and coitus, and predisposes to balanitis. The diagnosis of lesions of the glans penis such as chancre or carcinoma may be made difficult.

Treatment Circumcision under local anesthesia is recommended.

Paraphimosis

Paraphimosis may occur as a complication of phimosis. During intercourse or similar manipulations the narrowed opening of the foreskin may be pulled back behind the glans penis and cause obstruction of the circulation. Subsequently the glans becomes very edematous. Without treatment necrosis of the glans penis or, more often, of the narrowed prepuce, may occur.

Treatment If the paraphimosis cannot be reduced by manipulations surgical treatment (dorsal slit) is necessary and later on circumcision should be performed.

Peyronie's Disease (Induratio Penis Plastica, Penile Fibromatosis)

The disease consists of plaque-like fibrous proliferation and sclerosis of the corpora cavernosa with deformity of the penis. This leads to difficulties during intercourse. The condition is at times associated with Dupuytren's contracture. Good results may be obtained with x-ray or radium irradiation. Large amounts of vitamin E, female hormones and local injections of hydrocortisone also have been recommended.

Ulcus Vulvae Acutum (Lipschutz)

Coin sized ulcers are found on the inside of the vulva in young girls, sometimes accompanied by constitutional symptoms. Recovery usually occurs within a few weeks. *Bacillus crassus* is found abundantly in smears and has been considered the causative organism.

Treatment Application of antiseptic solutions or ointments (see page 58) is recommended.

Technique On normal skin a field 2.5 by 3.5 cm is marked with a blue skin pencil. One drop each of the sodium hydroxide and the phenolphthalein solution is dropped into the field and both drops are covered with a glass block. One measures the time until the red color disappears. In order to mix the liquid well the glass block is moved around every 30 seconds. The test is performed 10 times on the same skin site.

It takes 1 to 8 minutes for the color to disappear. The test is interpreted in the following way: 10 times decolorization at less than 5 minutes per test is considered good neutralization. A neutralization time of 5 to 7 minutes is considered average, and if the time needed for neutralization exceeds twice or several times 7 minutes this is considered slow neutralization.

Alkali Resistance

Technique Three adjacent fields 2 by 4 cm are marked with a skin pencil on normal skin. One drop ($\frac{1}{30}$ of 1 cc) of $\frac{1}{2}$ N sodium hydroxide solution is applied to each field. The drops are covered with a glass block. Ten minutes later the first drop is wiped off with cotton from the second and third fields and another drop of sodium hydroxide is applied. After 20 minutes a third drop is placed on the third field and is wiped off after 10 more minutes. If the alkali resistance is markedly diminished 1 drop suffices to produce small red nodules and blisters. If the resistance is only slightly diminished 2 drops cause such a reaction. Slight reactions following the application of 3 drops may be considered normal. If there is no reaction after application of 3 drops the alkali resistance is slightly increased. Papules, redness and small vesicles are seen immediately after the removal of the glass blocks but the reaction is more marked after 24 hours. At that time the reactions from alkali are transformed into small crusted erosions.

In about 10 to 20 per cent of the population the alkali resistance is more or less diminished and the time for alkali neutralization reduced. Persons with diminished alkali resistance are more susceptible to slight alkali damage of the skin. This is manifest in the form of dermatitis, paronychia and eczemas from cement, soaps and other alkaline agents.

Test for Spontaneous Rewarming of the Skin after a Cold Hand or Foot Bath

The temperature of the skin can be measured with thermoelectric elements or more simply with the thermomagnetic instrument of Heidenwolt. Changes of the skin temperature permit conclusions regarding the arterial circulation of the skin. The speed of rewarming

gives information

The test is carried

in the following manner: After the original temperature

advisable to perform intradermal tests only when scratch tests have been negative. Furthermore, one should not use too strong concentrations for the testing. Adrenaline (0.5 cc of a 1:1000 dilution subcutaneously) and intravenous antihistamines (see page 142) always should be ready for an emergency.

Tuberculin Test

The tuberculin test may be carried out in three forms:

1. In the Pirquet test a drop of undiluted old tuberculin (Koch) is applied to the skin, which is scraped slightly with the Pirquet drill by turning this instrument a few times. A control is carried out without tuberculin. A positive test is manifested by a red papule or pustule after 2 or 3 days.

2. Intradermal tests are carried out with diluted solutions of either purified extracts such as PPD (Parke-Davis) or old tuberculin. First an intradermal test with a freshly prepared solution of first strength PPD is carried out, if this test is negative for 48 hours second strength PPD may be used. The Mantoux test may also be performed with 0.1 cc of old tuberculin, starting with a dilution of 1:100,000 or 1:10,000. An infiltrated red papule within 24 to 48 hours indicates a positive result.

3. In the Vollmer patch test a specially prepared patch tape is applied for 48 hours and then removed and checked 48 hours later. A positive test is characterized by redness and infiltration at the site of the two outer patches and absence of reaction at the site of the control patch in the center.

Trichophyton Reaction

The technique corresponds to the Mantoux test. Dilutions of 1:50 to 1:10 are used at times even stronger concentrations.

Other skin tests with a similar technique are the Frei test in lymphogranuloma venereum, the Ito-Reenstierna reaction in chancroid, the Kveim test in sarcoidosis, and the Mitsuda test in leprosy.

Burckhardt's Test for Alkali Neutralization and Alkali Resistance

Alkali Neutralization

Reagents and materials

1. 1/80 N sodium hydroxide
2. Alcoholic phenolphthalein solution
3. Pipettes (30 drops per 1 cc)
4. Glass or Plexiglas blocks (2 by 1.5 by 3 cm)
5. Stopwatch

XIX. SELECTED COMMON CONTACT ALLERGENS AND THEIR PATCH TEST CONCENTRATIONS

Patch tests are easily interpreted when a high degree of allergic hypersensitivity exists. The eczematous reaction is caused by a dilution of the contact allergen much weaker than the concentration which causes a toxic irritant effect in normal persons. In dealing with an acute contact dermatitis following a relatively minor contact, it is better to start with the weaker concentrations and test stronger solutions only if the first tests are negative. However, it is always advisable to postpone patch testing until the acute phase has subsided in order to prevent unpleasant aggravations of the dermatitis. Recommended concentrations to be used in performing patch tests are listed in Tables 3 to 6.

Industrial Contactants

A considerable number of occupational dermatoses are due to factors other than allergic sensitization, for example, toxic irritant effects of the contactants. With alkaline agents especially diminished resistance of the skin plays a role, in these instances threshold concentrations of contactants may suffice to cause damage to the skin. Threshold concentrations are those dilutions which usually are still tolerated by normal skin, these are shown in Table 3. It is difficult to interpret positive reactions obtained with the use of threshold concentrations because they may also be positive in persons without dermatitis. In order to evaluate properly such positive patch tests in the causation of contact dermatitis, the clinical picture, the location of the dermatitis and the form of contact must be considered.

In cases of contact dermatitis from cement one should also check sensitivity to potassium dichromate, because cement contains traces of chromates.

has been established, a hand or a foot is immersed for 10 minutes in a bath of from 10 to 15° C. Afterwards the hand or foot is carefully dried, the temperature is measured every 3 minutes for a period of 20 minutes on the hand and 30 minutes on the feet. By this time, if there is good peripheral circulation, the level of the original temperature has been reached again completely or nearly so, if the circulation is poor, only an incomplete rewarming occurs. The room temperature should be 18 to 20° C and the test should be carried out on a non-fasting person with usual clothing. If the circulation is good the temperature reaches 25 to 30° C within 20 to 30 minutes. Rewarming is slowed down with a tendency to vascular contraction from cold.

The Thrombocyte Test

Ingestion or injection of the allergen causes a thrombocytopenia in allergic purpura, in several drug eruptions and in other allergic diseases. Counting the thrombocytes 30, 60 and 90 minutes after the ingestion or injection of the allergen may be used as a technique for discovering the allergen (Koller, Storck, Hoigne). Reduction of the thrombocytes to the extent of more than 30 per cent indicates an etiologic connection. The thrombocytes may be counted after the method of Fonio or with a phase microscope.

Medications

In most instances the concentration used for external treatment is suitable for patch tests. This, of course, does not apply to caustic or strong medications.

Table 4
Medications

Medicinal Contactant	Concentration for Patch tests
	%
Anesthetics (procaine, Novocain, Nupercaine, benzocaine, Surfacaine, Xylocaine)	1-5
Sulfonamides	1-5
Resorcin	1-3
Phenol	1-5
Penicillin procaine	1
Balsam of Peru	1-5
Ammoniated mercury and other mercurials	0.1-1
Lysol	5
Hilex	5
Quinine sulfate	1
Scarlet red	2
Vioform iodoform, Sterosan	1-3
Streptomycin	1
Tincture of iodine	As is, but without cover

Table 3
Industrial Contactants

Industrial Contactant	Concentration for Patch Test	Threshold Concentration
Turpentine	10-50 (in olive oil)	80-100
Turpentine containing waxes, floor waxes, shoe polish, paints	10-50 (in olive oil)	100
Gasoline, benzene	30-50 (in oil)	60-80
Cement, whitewash	10-20 (aqueous suspension)	40-50
Potassium dichromate	0.1-1	2
Chromic acid	1	
Nickel sulfate	2-10	25
Formalin (containing 40% formaldehyde)	2-10	50-100
Phenol	5	
Synthetic resins	As is, as powders	
Ammonium persulfate	5	
Photographic developer (hydroquinone, Metol)	5	
Soaps (toilet soaps, green soap)	1-2	30-100
Caustic soda	2	10-20
Cutting oil	30-50	
Alkaline detergents	1	5-10
Hydrochloric acid	1	5-10
Sulfuric acid	1	5-10
Sodium or potassium hydroxide	One half normal solution	
Dinitrochlorobenzol (explosives industry) and Trinitroanisol	0.1	

Plants

Where a high degree of sensitivity is suspected one may first try a short contact with the leaf before applying it as a patch test.

Table 6

Plants

Plant	Material for Patch Test
Rayweed and other weeds	Leaves with cover, or oleoresin extract from weeds (Graham Laboratories), without cover
African violets geraniums, chrysanthemum, Japanese primrose, other flowers	Leaf or flower
Poison ivy (<i>Rhus toxicodendron</i>) poison sumac poison oak	Leaves or oleoresin extract in acetone without cover or 1 1,000 dilution in oil (Caution!)
Oranges, lemons, potatoes, tomatoes, onions and other vegetables and fruits	Rind, applying both the outside and the inside

Clothing, Cosmetics and Dyes

Common contact allergens encountered in clothing, cosmetics and dyes are listed in Table 5

Table 5
Clothing, Cosmetics and Dyes

Contactant	Concentration for Patch Test
Paraphenylenediamine (dye for hair, fur, or clothing)	2% in Vaseline
Nail polish	Undiluted
Lipstick	Undiluted
Cosmetic ointments	Undiluted
Nickel containing jewelry (rings, earrings necklaces, bracelets, and watch bands)	Nickel coins or nickel sulfate, 2-10%
Garter clips containing nickel	As is, or nickel sulfate 2-10%
Clothing containing elastic (brassieres, girdles, garters)	Piece of elastic
Dress shields (containing rubber, formaldehyde and other deodorant substances)	Piece of dress shield
Hat band	As is
Dyed clothing material	Piece of material as is or moistened with 1% hydrochloric acid
Shoes	Piece of leather insole potassium dichromate, rubber, accelerators

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